New Orleans Medical

and

Surgical Journal

Vol. 83 . JANUARY, 1931 No. 7

THE OLD TIME COUNTRY DOCTOR AND TODAY'S SPECIALISM.*

GEORGE A. McBRIDE, M. D.,

JACKSON, MISS.

The span of a generation seemingly reaches back into antiquity, and we are not so much dwellers in a changed world as in an almost new world. Development has consisted of transformation, and evolution has been virtual revolution. Progress has proceeded by leaps and bounds! Radical has been the transition as pertaining to surgery and medicine. The old time doctor was surgeon, healer, executive—always ready for anything and everything. Early spring and the frost going out of the roads, nightfall and gusty, chilly drizzle rain. Plop!-plop!—a horse comes galloping through the mud. He pulls up before the doctor's office. In bursts the rider, and hastily announces that ten miles back in the lumber woods a man, just at the shutting-down hour, had gotten a hand badly mangled by a machine. Doctor and horse are soon off—two legs snugly tucked in a closely-curtained buggy, four legs bravely buffeting wind and rain, and sturdily o'er-mastering the heavy going.

From our reverie of the past we reawaken into a world electrified and gasolined. In this modern era automobiles and automobiles are everywhere. Roads are better and hospitals have become missionaries and pioneers. The hospital and the auto-car have sung the requiem, have tolled the knell of back-country surgery, which beyond dispute is all for the best.

We live and learn. Empirical knowledge is trustworthy knowledge. On earth we have many schoolmasters, but Grandsire Experience, faithful though stern, is lord over them all. Of his manifold lessons one of the most accentuated and most frequently repeated is caution in judgment—repression of every impulse toward jumping at conclusions.

The man or woman beset by the complications of 1929 is likely to cherish the dream of a time somewhat hazily known as the good old days, when living was simpler, less hurried, and far more endurable. Some things have improved and some gotten worse, according to the viewpoint. Try to imagine a few of the things that you use today that are the same in all respects as they were in your grandfather's day, and decide how many of them you would be content to live with. Some things are the same—chairs, knives, forks, plates—yes, yes, go on . . . but not matches, not razors, not underwear (thank heaven! not underwear), not lamps, not toothbrushes. Maybe hairbrushes, maybe shaving brushes—but a rag, a broom and a turkey wing are no more like a vacuum cleaner than the family album is like a movie show. We don't write with the same kind of pens. Typewriting is new. Our food is different. We can get up on wintry mornings to a house whose temperature has been regulated by a clock on the wall. Dinners are

*Read before the Central Medical Society, Jackson, May 20, 1930.

cooked on a stove that turns itself off when the roast is brown. The dishes are washed with a form of soap at which your grandmother would have rebelled, in a machine she could not understand, driven by a kind of power the existence of which she would have flatly denied. The sheer amount of drudgery that has been lifted out of living in the last fifty years justifies the phrase "Thank heavens!" that we might have used after our title and added "Gone are the good old days."

Our strength lies in focusing our energies and keenest intelligence upon a single point, and it may be possible to illuminate it as does the concentration of the sun rays upon any given point. Our weakness lies in thus concentrating and limiting our efforts, whereby we may possibly come to forget that we are physicians first and specialists after. The history of therapeutics is a history of blasted hopes. Where one new therapeutic agent has accomplished even a respectacle fraction of what was in the beginning claimed for it, a hundred, perhaps, have miserably failed. It is human nature to aspire eagerly to be the first, or among the first, to exploit the new.

In the rush for precedence conservatism is cast to the winds and, instead of waiting for a wider experience before giving expression as to the advantages and limitations of the new agent or method, we too often rush into print with the grossest exaggerations, with statements of therapeutic result that will not for a moment stand the test of a closer analysis. Honesty of purpose and absolute integrity in clinical reports constitute the one great shield and buckler of defence against the false and shallow optimism of the charlatan. Yet it is not enough that we be simply honest. Charlatans, it is true, are not generally honest, yet charlatanism is not inconsistent with personal integrity. Experience may be vast, integrity beyond question, but the basis of charlatanism is profound scientific ignorance. The charlatan never enunciates an idea, neither does he understand the principles underlying the cures that are wrought. He works blindly, and honestly believes that his simple, old-fashioned induction coil is equal to all sorts of impossible things, and well illustrates in his life and character the old truth that "the human mind follows the line of least resistance," believes what is easy and for personal interests to believe, and, except after the most careful training, cannot be trusted either to observe or to report its observations with accuracy. Combine, however, honesty of purpose with education and trained powers of observation and we have a lever which is capable of lifting any department of medicine to a plane of unquestioned honor and dignity.

There are those among us so short sighted as to think that instead of greeting the old style country doctor as a still useful member of the body politic, he should be given a valedictory. For some there are who mistakenly cherish the notion that the good old country practitioner is on his way out and that he shall soon disappear. Great is my desire to have it known that I am convinced that the general practitioner, the old family doctor who knows his people, their heredity, their proclivities, their possibilities, and who can gauge their psychological as well as their pathological content, is the one who should decide the question of specialization. Great office buildings filled too often with self-nominated specialists, technicians, laboratory men, all unmindful of the human appeal of the ailing—entirely alien to sympathy, to ideals to altruism.

Osler, who virtually laid down his life as a result of fatigues undergone in the interest of general practice, was a specialist, yes, but he had spent a novitiate in general practice under his model practitioner, James Bovell. Osler was a teacher who rose to the very pinnacle of such proficiency as was well known in his day but I feel quite sure that the great Osler would gladly subscribe to the dictum of

Dr. Frank Billings, himself a good specialist, who says quite simply and directly that "in the majority of cases the painstaking general practitioner has been able to make accurate diagnosis and to treat scientifically, and that too, without too expensive equipment."

All great advances bear within them the seeds of new problems. New methods of transportation expedite carrying of rural patients to urban centers. This is as it should be. Yet this fact has a demoralizing effect on the general practitioner who chooses the rural districts for location. Also, in these days of good roads, telephones, automobiles and airplanes, the larger centers can dispense urban service to rural communities.

I am old fashioned enough to believe that no man can properly and successfully specialize until he has spent at least five years in general practice. This experience gives any doctor a broader view and reduces the number of self-styled specialists to a minimum. I like to feel that a specialist is a limb of the great tree; body and roots of which are deeply grounded in all branches of human ailments.

The profession of medicine goes back to pre-Biblical days, and into remote lands and historical people. For instance, the Greeks had Esculapius, the father of medicine, who established some of the laws of health and treatment of disease. The evangelist Luke was a physician, receiving his education in Tarsus where there were masters of the profession, and near which stood the temple of Aesculapius, the first hospital ever established. Luke was thoroughly acquainted with the diseases he describes which he does in medical language of the times. We find Luke mentioning four cases, one, the son of the widow of Nain, the man with the dropsy, the ten lepers, and the woman bent with a spirit infirmity. There are other cases recorded by him of the other apostles. Indeed, the Good Samaritan's wounded patient is treated, according to Luke, by pouring oil and wine into his wounds. Indeed, Luke received a classical education, and he shows the mentality and characteristics of the cultivated Greek. We find him a learned and cultured doctor of his day, who typifies the right relation of the medical man to society. We find in him a supremely great man, and we of this day owe him a great debt.

Progress in medicine, since Luke's time, in which standards of preliminary as well as purely medical education have advanced so wonderfully, had its origin from within the medical profession. Neither lawyers or civil engineers or ministers advanced this idea, though possibly an occasional member of each or all these learned bodies may have thought higher education unnecessary for physicians. It is doubtful if any personal or local influence can turn the tide of the wide-sweeping movement for the finest standards of proficiency and skill, which will finally wipe out of existence the schools of the fake and the graft type which justly come under criticism.

While we are boasting over our cleverness and refinements over the ancients, how about the opinion of our patients. In former days the doctor was the most respected man in town. He was consulted for everything from A to Z. No one ever dreamed of doubting him. He was the hub of the joy, of the sorrow, of the business, of the pleasure, of the riches, and of the poverty of the community. What is he now? Has he mounted so high on his pedestal that he is above the notice of the people, or has he fallen off and gone so low that they ignore him? Why have the cults, quacks and charlatans gained such a foothold? Why are you asked, "Doctor of what?" when you make yourself known?

Why do patients consult you, then go to the chiropractor, Christian Scientist, or to their grandmother?

Empedocles said: ". . . so too in the body the good and healthy elements are to be indulged, and the bad elements and the elements of disease are not to be indulged but discouraged." And this is what the physician has to do, and in this the art of medicine consists: for medicine may be regarded generally as the knowledge of the loves and desires of the body and how to satisfy them or not; and the best physician is he who is able to separate fair love from foul, or to convert one into the other; and he who knows how to eradicate and how to implant love, whichever is required, and can reconcile the most hostile elements in the constitution and make them loving friends is a skillful practitioner." Out of mysticism, superstition and religious ritual the Greek went directly to nature and was the first to grasp the conception of medicine as an art based on accurate observation, and an integral part of the science of man. What could be more striking than the phrase in "The Law," "There are, in effect, two things, to know and to believe one who knows; to know is science, to believe one knows is ignorance. We are too prone today to listen to some smooth-talking, word-stuffed salesman and as a result, buy some new fangled contraption which he assures you will not only greatly impress your patient, and thereby enable you to ask a larger fee; but it will penetrate his remotest corner, diagnose the disease and cure the condition. Is this honest Medicine?

The unwillingness of young graduates to settle in country districts has been credited with partly accounting for the success of unlicensed medicine, and at least one great American physician has proposed a shortened medical curriculum for prospective country doctors. We must give this decreasing number of physicians in rural communities and the proposed remedies much thought. This is largely an economic problem. Better roads and automobiles have completely altered conditions of rural medical practice in recent years. The small town that kept two doctors busy in the old days can not longer support one, yet it is no worse served; for a doctor twenty miles away is as available today as the doctor two miles away ten years ago. The doctor must live and educate his children, and he cannot make a living in the small town, when all the best paying patients take their important cases to the larger towns twenty or thirty miles away. It is the country doctor, necessarily dependent on himself in all emergencies, who needs the most thorough training. Better roads, perhaps Henry Ford's airplanes will solve the problem of country practice. And soon, I trust, there will be small rural hospitals, with out-patients departments for rural communities. In the meantime the problem is a serious one, and not to be solved by cheap medical education. Not that I think medical education is all it ought to be; the truth is, the educational necessities of modern practice have become too big. Medicine, long empirical, is coming into the scientific inheritance of the last twenty years of hitherto undreamed of increase of accurate knowledge, and to be abreast of scientific advance we must practice scientifically exact methods. This is almost impossible without laboratory and hospital facilities. Nay, more, our curricula are so overcrowded that the student has no time for intelligent thinking in college, and is too tired for effective home study at night. Teachers are more troubled than students over the difficulty, and I have no remedy to offer.

It has been said that you are a real country doctor if you can change tires at four below at four a. m.; if you can set a fractured femur with a piece of string and a flat iron and get as good results as the mechanical engineering staff of a city hospital at ten percent of their fee; if you can drive through ten miles of mud to ease the little child of a deadbeat; if you can do a podalic version on the kitchen table of a farm house with husband holding legs and grandma giving chloroform; if you can diagnose tonsillitis from diph-

theria with a laboratory forty-eight hours away; if you can pull the three-pronged fishhook molar of the two hundred fifty pound hired man; if you can maintain your equilibrium when the lordly specialist sneeringly refers to the general practitioner.

Some reversion to the old preceptor method is indicated in order to keep the medical students' perspective always true to the lode-star of ethical medicine. In the premedical work, if anything should be eliminated, I would say eliminate the requirement regarding a smattering of modern languages; it never gets anywhere. Solidity, not veneer is wanted. In our medical schools today, it is unfortunate that our students do not come in contact with practicing physicians until their Junior year. Teachers must be sought whose vision is as broad as their training must be deep. Here again, unfortunately, the teachers are too often full-time specialists. Some of the greatest teachers in the history of medicine in this country, such as Billings, Senn, Murphy, Quin, Fenger, Davis, all specialists in their later lives, trained to the task in great fields of medical practice, were the type that today are too infrequent. These men taught scientific medicine, altruism and devotion to humanity, and they kept alive in the student mind, an awareness of current conditions and of what should be expected of every doctor as the priest of the sacred human body. The practical things in medicine should not be suppressed at the expense of scientific medicine. Too many men trained according to the present tendency, bloom forth as specialists and lack the training necessary to endure and sacrifice.

Our refuge, our help, must come through the rearing of the medical men on the old ethical lines, close contact throughout the student years with the men of general practice, and if possible, by extending the arm of fellowship from the State Society in a protecting and encouraging way to the student. We must have cooperation; mutual sympathy and respect. We must present a united front against all that is unethical, shifty, questionable, or merely expedient. We must recover the control of medical education and medical practice by medical men.

We close with the words of the eminent writer and teacher, Hobart A. Hare of Philadelphia:

The endeavor of the true physician is "To cure sometimes, to relieve often; to comfort always".

SURGERY OF THE OVARY.*

MAURICE J. GELPI, M. D.,

NEW ORLEANS.

In considering a subject in the light of experience, one's attention is often arrested by certain impressions which stand out in comparatively bold relief. Stimulated by such observations, I shall speak here, though briefly, on certain diagnostic and symptomatic phases of ovarian surgery as well as on certain pathological and technical considerations.

The topics of diagnosis and symptomatology are so closely interwoven that they lend themselves readily to simultaneous discussion. Everyone is familiar with the importance of differentiating ovarian pain from that produced by such classical disturbances as ureteral stone, infection along the genito-urinary tract and tubal pregnancy. The means at our command capable of assisting us in determining the existence of these conditions as well as the measures available for combating them are also familiar to everyone. There is a certain pathology, however, which for a number of years I had completely overlooked as being capable of simulating ovarian pain. I am referring here to disease of the descending colon, sigmoid and recto-sigmoid. In the last few years I have been very much impressed by the

*Read before the Louisiana State Medical Society, Shreveport, April 29, May 1, 1930.

number of women who complained of low, left-sided pain very closely resembling that produced by tubo-ovarian diseases, who were really suffering from colitis or sigmoiditis. Occasionally such patients were encountered who had already been operated upon for supposed pelvic disease and who were never relieved until the pathology in the colon was discovered and treated. Such a patient was recently seen who had not only been operated upon without relief, but was about to undergo a second exploratory pelvic operation, when the colonic disturbance was discovered and relief brought about by properly directed attention. My own experience makes me feel that no doubt in the past, many an unoffending ovary has thus been needlessly sacrificed. The laboratory and particularly the roentgen-ray is of decided assistance from a diagnostic standpoint, keeping in mind the value of the barium enema for the roentgenologic study of the colon.

As regards pathology and surgical technic, certain points come to mind which might stimulate discussion. It might be recalled here that the handling of malignant ovarian neoplasms calls for the same radicalism that malignancy elsewhere requires, irrespective of age. But while in ovarian malignancy total extirpation not only of the primary growth, but of the adjoining internal genitalia would appear to be, when feasable, the wisest procedure, we see in contrast to this, the necessity of ovarian conservation in all conditions except malignant and non-malignant neoplasms and ovarian tuberculosis. This refers particularly of course to women far from menopause. I learned some years ago, from Dr. Hurdon at Johns Hopkins, when medical scientists of the gentler sex were not so numerous as they are today, some very interesting facts of practical value. In early tubal tuberculosis, the ovary is often not involved, and when it is, the pathology is usually plainly discernible on the surface. When an element of doubt arises, an incision will permit of opening the ovary like a book affording a further means of inspection when there are no discernable lesions superficially. The practical application of this is that by this means, in the case of young women, often complete ovarian conservatism can be and should be practiced. In the case of early tubal tuberculosis, this is particularly desirable, not only to avoid a premature and precipitate menopause, but also because after removal of the primary tubal focus, the accompanying endometrial tuberculosis apparently improves with each succeeding menstrual period and finally disappears completely. I shall say while on the subject of ovarian conservation just a few words in this regard, in pelvic infections affecting young women.

I am convinced, from observation of the young, neurological, oophorectomised wrecks, only too often encountered, that in advanced pelvic inflammatory disease, in young women, it is best whenever possible, to leave in a piece of doubtful ovary, even though it might subsequently give trouble. This is much better practice than to ruthlessly remove every vestige of ovarian tissue. This is particularly true in the case of Neisserian infections where the late pathology, so far as the internal genitals are concerned, is always principally in the tubes. This suggests the subject of ovarian transplantations.

In my hands ovarian transplantation to distant parts of the body has been most disappointing. Furthermore I could never understand the logic of removing an ovary or piece of ovary from its normal habitat to transplant it in foreign surroundings. If the ovary, either in whole or in part, is sufficiently good to transplant, then it would seem more reasonable not to transplant it at all, but rather to leave it where it is and where it belongs. It would seem more likely to function in its normal surroundings. Furthermore, better circulation is more likely obtainable in its natural environment. Of course, I am referring practicularly here to autogenous grafts. The last topic I shall discuss is purely technical.

The more I study pelvic pathology, the more difficult I find it in the exceptional case, to differentiate every time in the gross between the simple cyst-adenoma and the potentially malignant papillary cyst-adenoma. For this reason I have come to the conclusion that it is far safer not to tap ovarian cysts. The danger of grafting an unsuspected papillary cyst is by this means positively avoided. It is safer therefore, even in the case of very large tumors, to make a sufficiently generous incision to deliver the cyst without releasing the contents. I feel quite certain that many will be ready to challenge this attitude and yet from the standpoint of safety in technic, my observations I believe will stand the closest scrutiny.

The points which I have tried to emphasize without presuming too much on your patience are these:

1. That before contemplating surgical attack on the ovary, we should make use of every reasonable means at our command, to first determine that the ovary should really be our objective. In this connection attention was called to the simulation of ovarian pain which may be produced by disease of the colon, sigmoid and recto-sigmoid.

2. That while ovarian malignancy calls for radicalism irrespective of age, conservatism should be the keynote in all other conditions not neoplastic and not ovarian tuberculosis. The latter applies particularly to the treatment of young women.

3. That in early tubal tuberculosis, in addition to avoiding an early, precipitate menopause, ovarian conservation is a useful factor in eliminating endometrial tuberculosis after removal of the focus in the tubes.

4. That it is worth while to preserve some ovarian function in young women suffering from pelvic inflammatory disease, even though the ovary or piece of ovary preserved might possibly give trouble at some future time.

5. That leaving in an ovary or part of an ovary would seem to be more logical than transplantation to distant parts of the body.

6. Finally, that it would seem to be safer and therefore better technic not to tap ovarian cysts, but rather to remove them through an adequate incision.

DISCUSSIONS.

Dr. Levy (New Orleans): I am sorry Dr. Crawford is not here. With his great fund of experience, he would have given you a much more interesting discussion, I am sure, but it is not difficult to discuss the surgery of the ovary after hearing such an elucidating paper as you have just heard.

"Surgery of the pelvis," Dr. Will Mayo said some years ago, "is the great delusion of surgical mortality." A great many surgeons have been tackling—and getting away with—surgery of the pelvic organs without the high mortality, because the pelvis can stand so much. The pelvis has a great amount of resistance and your mortality is lower. When you get in the upper abdomen, you have to be a little bit better technician than in the lower abdomen, and your mortality is lower in the pelvis though the morbidity might be higher.

The Removal of the Ovary. We have stressed and stressed about leaving ovaries, and rightly so. Dr. Gelpi insists upon it, and it is the right thing to do in spite of what the gland hounds tell us. We all know of the harrowing experiences we have when we take out ovaries.

Resection of Ovaries. An ovary is either good enough to stay in or bad enough to come out, provided you can have a fairly good ovary on one side.

Do Not Tap Cysts. Just for the reason that Dr. Gelpi emphasized. You might get a carcinoma, malignant ovary, and just spread seeds on a fertile field, and have a general carcinomatosis before you know it. A great many of these malignant ovaries can be taken out, if you will just take them out toto without spilling the contents, but if you spill the contents, then you have spread seeds on a fertile field and you may expect a general carcinomatosis to rapidly follow.

A great many pelvic infections will clear up if let alone. Rest, and plenty of it, and a great deal of surgery of the ovary can be avoided if you will just stay out of the abdomen of that type. It was a very common thing a few years ago to go

into the pelvis and clean it up. Several years ago, we left only one ovary and that was enough, but now we can leave a lot of pelvic organs and still have good results. It is better, however, in ovarian graft, if the disease is near the pedicle of the ovary and you have to graft an ovary, it is better to take particles of both ovaries and transplant them than to have no ovary at all. Some men have gone so far as to claim that they have even seen menstruation after a grafted ovary. We know though that it is so important to conserve the ovary that we are willing to graft it whenever feasible rather than to remove the entire organ.

Again I want to thank Dr. Gelpi for his paper and to apologize for this impromptu criticism.

Dr. H. W. Kostmayer (New Orleans): I want to talk on just one phase of this very big subject that has been so nicely covered and that apparently hasn't been given consideration, and that is the feasibility of too many operations on one patient.

In lecturing to medical students and to nurses during a great many years, I have always told them that if each one of them could have a laparotomy performed on themselves, they would have a great deal more consideration for the patient whose abdomen has been opened. I think this might be true of surgeons in middle life. Having been through the surgical mill so many times myself, I have a great fondness for making a patient avoid a second operation if I possibly can. I think that ultraconservatism can be practiced and during many years of gynecological work, I have had some very keen regrets for having left in diseased ovarian tissue. So, I think one ought to be pretty certain in leaving in an ovary, or piece of ovary, that it is going to function and not going to have to be removed in a second operation.

I think the reason for transplanting ovarian tissue from its natural habitat, where there is a question in the mind of the surgeon, is to make it more accessible for future removal. And an added reason is to take it out of a diseased atmosphere and to put it into a healthy one.

I rose merely to urge that while we want to be exceedingly conservative with female sex organs, we do not want to be ultraconservative and necessitate a second operation. Here we see the results of good judgment which can only come after many years of experience in this line.

Dr. T. B. Sellers (New Orleans): No doubt there are too many ovaries being sacrificed in the hands of the average surgeon. I am sure that Dr. Gelpi does not mean to imply that there is any harm in puncturing an ordinary follicular cyst of the ovary. I agree with him that there is danger in puncturing the true ovarian cyst on account of the possibility of spreading malignancy.

I want to make a plea for conservative ovarian surgery. In several cases, I have resected a portion of one ovary and later opened the abdomen for some other condition and found that the resected ovary was the only ovarian tissue that I could conserve. However, we must not lose sight of the fact that our standard of conservatism is much lower in women of the working class than in those who are able to lead a more sedentary life following operative procedures. This point has been forcefully impressed upon the men who have handled large Charity Hospital services.

Dr. T. J. Fleming (New Orleans): I certainly enjoyed Dr. Gelpi's paper.

One question: What is his opinion on plastic surgery of the ovary? And in regard to transplanting ovaries, I have tried transplanting ovaries a few times and have had to go back and take them out. I believe if an ovary is worth anything at all, after transplanting, it would be worth a lot more left where the normal blood supply is and in its normal position.

Dr. Gelpi (closing): I find it very difficult in discussing certain surgical topics to succeed in not being too dogmatic and yet carry a point. Very often one is inclined to say, "This is 100 per cent so," when, as a matter of fact, deep down in your heart, you know it is only 99 per cent so. But you are more likely to carry a point if you say 100 per cent.

In regard to Dr. Levy's discussion on resection, and that will also answer the last speaker's question, I believe—I am frank to confess—that the more I go into this work, the fewer resections of ovaries I feel it necessary to do. There, again, we can't be too dogmatic and we can't say you must never resect an ovary, but I feel that the majority of gynecologists feel like I do—that there are fewer ovaries resected today than there were in the past. The reason is that everybody knows the ordinary cyst does not need any treatment.

In regard to Dr. Kostmayer, I think he struck the key-note of the whole situation when he mentioned the word "judgment." If you are handling pelvic pathology, in certain types of women, you are going to be influenced to a great extent by the economic situation, which Dr. Sellers also brought up. You are not going to treat a marked, advanced pelvic case in a colored servant in the same way you would treat a very fine lady with the same condition, who can afford the long rest

in bed, preoperative care, etc., that these people can afford. There, as I say, the whole question resolves itself into the question of judgment.

As regards Dr. Sellers, I think I have already mentioned the economic situation that really resolves itself into the question I have just discussed.

I want to thank you gentlemen for discussing my paper.

UPPER LEFT ABDOMINAL TUMOR MASS IN CHILDREN AND SOME POINTS IN DIFFERENTIAL DIAGNOSIS.*

S. L. BRISTER, JR., M. D.,

GREENWOOD, MISS.

In this paper I will discuss tumor mass in the left upper quadrant in children, paying particular attention to enlargements of the spleen and kidney, the blood pictures produced by each, and some differential diagnostic points. Of the organs found in this quadrant the spleen and the kidney are the most important ones, and of these two the spleen holds the spotlight. Of the diseases to be considered, some are more interesting than others, due to the fact that some are very rare. Many of us may never see some of these conditions, but tomorrow as we go back to our work, some of us will be confronted with these conditions, because they are going to be and it falls the lot of some of us here, to make the diagnosis.

Of the diseases to be discussed that produce enlargement of the spleen are certain blood dyscrasias as:

1. The leukemias, both the lymphatic and the myelogenous.
2. Banti's Disease.
3. Hodgkin's Disease.
4. Gaucher's Disease.
5. Familial jaundice.
6. Von Jaksch's anemia.

Certain malignant tumors of the spleen and the kidney as:

1. Lympho-sarcoma of the spleen or kidney.
2. Hypernephroma of the kidney.
3. Congenital polycystic kidney.
4. Hydronephrosis of the kidney.

Certain acute and chronic infections causing enlargement of the spleen, the most important of which are:

1. Typhoid Fever.
2. Malaria.
3. Syphilis.
4. Tuberculosis.

On physical examination a mass is felt in the upper left quadrant; is it kidney or spleen? Sometimes it is rather difficult to distinguish between the two. The outstanding differential points are as follows: In the splenic enlargement, you feel a flat oval mass lying in close relation to the diaphragm, at the level of the ninth and tenth rib, and above the kidney. There is a definite notch in the spleen that can be felt. In percussion the note is dull indicating that the spleen is close to the anterior abdominal wall. In the splenic enlargements, the blood picture is very essential in deteriming whether you are dealing with spleen or kidney mass. The blood count will show characteristic changes and in the kidney conditions, the picture is not characteristic.

In the cases of kidney mass, the important differential points are that the left kidney lies to the side of the spine, on the muscular bed formed by the lumbar muscles at the level of the eleventh and twelfth ribs. This shows that the kidney lies closer to the posterior abdominal wall, is below the level of the spleen, posterior to the colon, stomach, and intestines, which will give you a tympanitic percussion note over the kidney area from the anterior surface. The kidney moves slightly with respiration. There is no evidence of the notch. The blood picture reveals only a secondary anemia, but the urine examination shows that there is definite trouble in the kidney. Of the differen-

---

*Read before the Section on Medicine at the Sixty-third Annual Session of the Mississippi State Medical Association, Vicksburg, May 14, 1930.

tial points discussed above, too much strees as to the importance of a blood and urine examination cannot be made.

Now, considering the diseases themselves, let us first consider the leukemias. There are two types, the lymphatic and the myelogenous. The lymphatic is characterized by slight splenic enlargement, slight glandular enlargement, marked leukocytosis, slight anemia, the differential count showing a marked increase in the lymphocytes usually the small ones, which may be in proportion as high as 90 per cent.

In the myelogenous type the splenic enlargement is much greater, the glandular enlargement is greater, the anemia is of the pernicious type, there is marked leukocytosis, the predominating cell being the myelocyte. The liver in both types is enlarged, but to greater proportions in the myelogenous type.

Banti's disease is characterized by splenic enlargement associated with hepatic cirrhosis. This condition does not show the marked glandular involvement of Hodgkins' disease, or the peculiar blood changes of the leukemias, but presents a picture of marked and progressive anemia, persistent leukopenia, increased amount of urobilin in the urine, and a marked degree of jaundice.

Hodgkin's disease presents a picture of marked general glandular involvement usually beginning in the cervical region, and splenic enlargement. The blood examination shows a secondary anemia and definite and marked increase in eosinophils. There are skin manifestations such as pruritis, and general itching of the skin. Biopsy of one of the glands will help clear up the diagnosis as it gives a definite picture.

Gaucher's disease is a familial, but not hereditary, disease and also produces an enlargement of the spleen. It is characterized by a peculiar brownish pigmentation of the skin, especially of the face, slight anemia, the lymph glands may be only slightly enlarged, leukopenia not so marked, differential count within the normal limits. Sometimes there is a yellowish wedge-shaped thickening of the conjunctival on either side of the cornea.

Hemolytic jaundice is a congenital and hereditary disease characterized by a mild or marked degree of jaundice, moderate anemia, lymph glands not enlarged, spleen markedly enlarged, liver normal or only slightly enlarged, reticulated red blood cells predominating the field in proportion of 25 to 50 per cent, a definite increase in the fragility of the red cells, and an increase in the urobilin content of the urine.

Von Jaksch's anemia, or pseudo-leukemic anemia causes a definite enlargement of the spleen and is a disease of the first two years of life. It is associated with marked rickets, high grade anemia, moderately enlarged liver, very low hemoglobin percentage, marked leukocytosis, and the lymph glands not, or only slightly palpable.

Now that we have finished with the most important blood dyscrasias, let us pass on to the tumors of the kidney and the spleen. In tumors of the kidney, almost all of them are malignant. There are several varieties of tumors, such as lympho-sarcoma, hypernephroma, which is a malignancy of the suparenal gland of the kidney, congenital polycystic kidney, and hydronephrosis. The diagnosis of the types of tumor is rather difficult before autopsy or operation, but the important things to remember are that the tumor mass is present, hematuria,

marked cachexia, secondary anemia, patient usually under five years of age, and that the blood picture is relatively unchanged.

Tumors of the spleen are very rare. They may be primary or secondary, usually secondary, and of the lymphosarcoma variety. If the lymph glands are involved, a biopsy will help clear up the diagnosis. Examinations of the blood will show only a mild anemia, and none of the characteristic changes of the blood dyscrasias with an enlargement of the spleen. The urine examination shows none of the characteristic change of the tumors or disease of the kidney.

Of the acute and chronic infections which will cause an enlargement of the spleen, the most important ones to be considered are typhoid fever, malaria, tuberculosis, and luetic conditions. Typhoid fever gives an enlargement of the spleen, normal blood picture or a mild anemia and slight leukopenia, differential count within normal limits except for maybe a slight increase in the number of lymphocytes. The diagnosis is made on the strength of the clinical picture with a positive Widal test or a positive blood culture, or both.

In malaria, the spleen is enlarged, and sometimes to a marked degree, the blood picture is within the normal except in some cases there may be a mild anemia, slight leukopenia, and the differential count is normal. A positive diagnosis is made with the clinical picture and with the demonstration of the malarial parasite in the blood smear. A negative smear does not mean that your patient does not have malaria and in such cases the history, symptoms, and physical findings must be relied upon, and the therapeutic test tried. If the patient clears up, the case is more than likely one of malaria, due to the fact that quinine has very little effect upon the other types of fever.

Tuberculosis will cause an enlargement of the spleen and the history, symptoms, physical findings, roentgen-ray pictures, sputum examination, tuberculin test, and the blood examination will help in establishing a diagnosis. The blood picture shows only slight anemia, normal or only a slight reduction of the white cells, and the differential count is normal, or there may be a slight increase in the number of lymphocytes. Biopsy, if glandular involvement is present, will help clear up the diagnosis.

Syphilitic infections will cause an enlargement of the spleen, and the diagnosis is made by the history, symptoms, physical signs, and a positive Wassermann test. The blood is within the normal limits. There is a generalized enlargement of the lymph glands from a mild to a marked degree.

In conclusion, I want to emphasize the importance of the laboratory findings in clearing up the diagnosis in dealing with the enlargements in the upper left abdominal quadrant. The blood pictures will clear up the splenic involvements, and the urine examination will clear up the kidney involvements.

DISCUSSION.

Dr. S. L. Brister: There is very little to say about treatment of these cases. Usually it should be surgical treatment as early as diagnosis is made. Surgical removal of the spleen or kidney should be done as soon as pathology is diagnosed. It may be necessary to transfuse the patient or build them up before they will stand operation. Be sure the other kidney is not involved by metatasis or some other condition. Roentgen-ray should be made of the chest, the long bones, and the spleen. The most promising results are obtained from surgical procedure.

MENINGOCOCCEMIA FOR EIGHT MONTHS FOLLOWING MENINGITIS: RECOVERY.*

I. I. LEMANN, M. D.,†

NEW ORLEANS,

AND

H. E. TEASLEY, M. D.,‡

ATLANTA, GA.

Since Gwyn's[1] report in 1898 of septicemia with meningococci a very considerable literature has accumulated on the subject. It has in the meantime become generally recognized that Herrick's [2] contention is correct: meningococcal meningitis should be regarded as essentially a septicemia with meningeal localization. Of particular interest, especially from the standpoint of diagnosis, is the group of cases of chronic meningococcal septicemia in which the meningeal localization was either absent altogether (so-called "pure" septicemia) or was delayed or in still other cases recurred a number of times. To this group belongs the case which we are about to relate where the septicemia continued for eight months after the cure of the meningitis. Soloman[3] in 1902 was the first to direct attention to this group. William Dock[4] in 1924 reporting a case of "intermittent fever seven months duration due to meningococcemia" analyzed 68 previously reported cases. Marlow[5] last year remarks that 13 cases have been published in the American literature and that the foreign literature contains reports of approximately 110 cases. Graves, Dulaney and Michelson[6] in 1929 give a list of 13 cases in the American literature to which is to be added Marlow's case, that of Vesell and Barsky[7] and that of Moleen and Seecof[8] and the present case. This makes a total in the American literature now of 17. Those who are interested will find an excellent bibliography in the report of Morgan[9] which has been brought up to date by additions in the reports of Graves, et al,[6] Marlow[5] and Vesell and Barsky.[7]

*Read before the Orleans Parish Medical Society, June 9, 1930.

†From the Medical Service, Touro Infirmary.

‡From the Pathological Department, Touro Infirmary.

CASE REPORT.

Miss L. P., aged 23 years, pupil nurse, was admitted December 2, 1928, 9:30 p. m. At that time there was a prevailing small epidemic of "flu" among the student nurses. The patient had been complaining for the past few hours of severe headache and nausea. The headaches continued very severe. The patient developed great restlessness and photophobia. Lumbar puncture yielded cloudy fluid under pressure, showing meningococci. Temperature curve varied between 100°-102°. Antimeningococcic serum was given intraspinally and intravenously repeatedly between December 5, 1928 and December 12, 1928. The serum used at first was that of manufacturer A, later preparations of manufacturer B and of manufacturer C were used. The reason for this shift was that after no decided improvement resulted from the use of serums A and B it was found that serum A agglutinated the organisms from the patient's spinal fluid only in dilutions of 1 to 10, serum B in dilutions 1 to 30 but serum C in dilutions up to 1 to 80. Although it is not definitely known that the agglutinating power of a serum is any index of its potency, still it seemed worth while making a trial on this basis. Clinically there seemed to be reason to believe that serum C was the most potent. She entered into convalesence December 19, 1928 and after this time the temperature never rose above 99° except on January 31 when it rose abruptly to 104° and just as abruptly fell to normal. She was finally discharged on February 9, 1929 apparently in very good condition and was sent to her home about 100 miles away. Throat cultures at that time were negative.

She was readmitted on April 12, 1929. She said that two days before she left the hospital she had a chill and another one four days after that. Since then she had had chills at irregular intervals from one to two weeks apart. She would break out with a number of red, flat spots and then would have a chill lasting four or five hours with generalized aching. The temperature would rise to 103°-104° and then drop rather rapidly. After this she would have many urticarial wheals which would last from one to two days. In the intervals between these chills she would feel very well. She had not been confined to bed, in fact she had been going about attending dances, etc.

The physical examination was made soon after a chill. She was still crying with discomfort and

general malaise. She was well nourished and well oriented. There were a number of isolated red spots varying from ½ to 1 cm. in diameter, most of them showing a pin point to slightly larger deeper red central area. Some of the lesions were slightly elevated, some of them slightly painful. There were no lesions on the feet nor toes; nor was there any history of any having occurred there. The patient said she had had some on the palms and soles. On the external aspect of the right knee there was an old discolored ecchymosis which the patient claimed was due to a blow. Over the anterior surface of the tibia were similar greenish areas. There had been no blows here. No adenopathy. Mucous membranes: Normal. Teeth: Excellent. No tenderness over any of the paranasal sinuses. Pharynx: Negative. Neck: Negative. Heart and lungs: Normal. Abdomen: Vague tenderness on palpation in the right hypochondrium. Definite tenderness in right lumbar region (kidney fossa). Liver and spleen not felt. There was some tenderness over both hips but no pain was elicited by moving the extremities in any position in any direction. The patient was well oriented as already indicated and gave a clear account of the interval since her former illness. There was no indication of any meningeal irritation; no photophobia, no Kernig, etc. Roentgen-ray studies of the paranasal sinuses and of the hip region yielded no information.

The day after her admission she had a crop of definite urticarial wheals which itched greatly and were very different from the red, painful spots already described. An intra-dermal test with horse serum was made a day after the disappearance of the urticarial wheals. This produced characteristic positive reaction. Dermal tests with food stuffs and pollens yielded negative results.

From April 12 to 28 there were eight sharp elevations of temperature, at first between 102°-103° and later ranging from 100°-101°. Then followed a period of calm until May 11 when the temperature rose abruptly to 102.6°, falling at once. Again the temperature was normal for a week. On May 18 and 20 slight transitory elevations of temperature, 99.5°-100°. On May 26 the temperature rose to 102.4°, falling at once. Again a period of calm until June 2 when the temperature rose to 103.6°. The patient was again apparently perfectly well until June 19 when there was another chill and rise of temperature to 104.2°. On June 23 there was a rise to 101°. All of these rises were abrupt followed by an abrupt fall. On June 26 the temperature began to rise gradually, reaching its maximum 102° on June 27, twenty-four hours later. This in turn was followed by an abrupt fall. On June 29 there was a slight elevation of temperature, about 100°. From July 9 to 11 there was an irregular elevation of temperature of 99.6°-100.4°. On July 14, again an elevation to 99.5°. On July 17, an elevation to 99.6°. There were again abrupt rises on July 24 to 102.4° and on July 27 to 103°. The rest of the temperature curve was flat.

She was finally discharged August 19 after she had been normal for twenty-three days. She has continued to be perfectly well and without any fever and without any return of the eruption or joint pains to this date, May 21, 1930.

The progress record shows that she continued to have crops of red spots, usually at the time of the chill and elevations of temperature though she would have at times these spots without the paroxysm. The red spots would occur any where upon the body or limbs. Sometimes they would be upon the fingers, palms, soles; at other times along the limbs, upper and lower, sometimes upon the trunk. At one time she had considerable pain and tenderness in the abdomen simulating an acute intra-abdominal surgical condition and this was attributed to the localization of one of the emboli in the abdominal wall. From time to time she had complained of aching and pain in various joints—hips, knees, elbows and shoulders. At these times active movements were painful, passive movements were not. At no time was there redness, heat nor swelling over the joints.

Once she complained of "pain around the heart" but this was slight and very transitory. At no time was there tenderness on percussion over the sternum. The heart fortunately was never involved. The urine showed no abnormalities. The spleen was noted as being palpable once at the beginning of July, otherwise repeatedly the note is made that the spleen was not felt..

We wish to emphasize that throughout, the general condition and nutrition was excellent. There was never at any time, even during the paroxysms, any phenomena of toxemia. Her spirits and morale were good except when she was suffering with acute pain and when she was bored by the tedium of her confinement. She finally rebelled because of this latter and protested against her long imprisonment. The picture, however, throughout was that of a well nourished, bright, vivacious, thoroughly interested young woman without any objective evidence of anemia.

Her blood picture on admission on April 12, 1929 was:

Red blood cells.....3,900,000
White blood cells..... 6,800

Hemoglobin 80 per cent

Polys .....81 per cent
Lym. .....18 " "
Eos. ..... 1 " "

At one time at the end of June her red blood cells were reported as low as 3,305,000. At this time the rest of the blood picture was:

White blood cells..... 8,750

Hemoglobin 80 per cent

Polys .....81 per cent
L. M. ..... 5 " "
S. M. .....12 " "
Eos. ..... 2 " "

The highest leukocyte count was 12,000 on June 5 and the lowest was 4,000 on May 28. At her discharge on August 18 the blood picture was:

Red blood cells.....4,950,000
White blood cells..... 5,750

Hemoglobin 80 per cent

Polys .....50 per cent
L. M. ..... 6 " "
S. M. .....44 " "

Blood cultures were as follows:

4-12-29 Staphylococcus aureus showing hemolysis in bouillon but not in plate. Negative after 72 hours.
4-14-29 Negative.
4-17-29 Negative.
4-19-29 Staphylococcus aureus in bouillon but not in plates.
4-23-20 Gram negative diplococcus (culturally found to be meningococcus).
4-28-29 Negative.
5-11-29 Negative.
5-25-29 Negative.
5-26-29 Negative.
5-26-29 Negative.
5-30-29 Negative.
6-19-29 Gram negative diplococcus culturally meningococcus.
6-19-29 Negative.
6-29-29 Negative.

These cultures were made in:

1. Bouillon slightly alkaline to phenolphthalein to which was added 1 per cent dextrose. Approximately 4 c.c. of blood to 100 c.c. medium were used.

2. Agar which was prepared as above except that 2 per cent agar was used and no dextrose.

Positive cultures were obtained only on the bouillon cultures and not on the plates. The organisms would grow when transplanted to the surface of the plates or to blood agar slants. These sub-cultures had to be transplanted each day and had to be kept at incubator temperature; if this were not done the transplants would show no growth. After the first positive culture, the only positive cultures were obtained when the blood was taken very early in the rise of the temperature. When the blood was taken at the height of the temperature no growth was obtained.

Cultures of the throat and naso-pharynx were negative throughout. They were made frequently. Throat cultures in her original illness and for some weeks after recovery had shown the meningococcus as the predominating organism.

A lumbar puncture was not done for reasons which we shall discuss later.

Therapy: There was evidence that the patient was very sensitive to horse serum. We did not venture to give her any meningococcic serum. Vaccines prepared from cultures from her own blood were given, as follows:

June 9, 1929.....250 million
June 14, 1929.....500 million

They were then discontinued because the patient had sharp paroxysms of chills, fever and discomfort. Injections were not resumed until July 27 when she received 750 million. Further injections were continued, as follows:

July 31, 1929.....1 billion
August 4 1929.....1 billion
August 9, 1929.....1 billion
August 19, 1929.....1 billion

These injections were continued even after the patient left the hospital and until the end of September.

COMMENT.

There are several features of this patient's illness to which we desire to call attention. The family history is quite remarkable. In 1923 a sister of the patient, at that time also a young woman, died at Touro Infirmary with meningococcus meningitis in the service of Dr. C. L. Eshleman who has been kind enough to furnish us with some notes of the case. Spinal fluid showed meningococcus. The blood culture was negative. Nine years before, in 1914, a brother had died. The onset of his illness had been very similar to that

of Dr. Eshleman's patient. He had fever and headache and occasional vomiting for about three weeks before he was brought to New Orleans where he was seen at the Touro Infirmary by Dr. J. B. Elliott by whom he was transferred to Dr. R. M. Van Wart. The patient was delirious at times with moderate fever. At other times the temperature was normal and the patient was more rational. He vomited at times. Blood cultures and spinal punctures threw no light on the condition. Two small doses of anti-meningococcic serum were given intraspinally. After two months Dr. R. Matas did a craniotomy. Fluid from the ventricles showed meningococcus. The patient died five or six days after the operation. Here then is a remarkable series of events which can hardly be regarded as accidental. In the same family there was a meningococcus infection in 1914, 1923 and 1929. Are we to believe that some member of the family continued to be a meningococcus carrier throughout fifteen years? Can we on the other hand explain the remarkable coincidences by assuming that the brother and two sisters were peculiarly susceptible to attack by the meningococcus and that each one in turn when exposed fell victim? In either event we are confronted with an interesting problem in public health. On the other hand it would seem improbable that our patient was a source of danger to others during the period of her septicemia when the cultures from her nose and throat were always negative. It must be granted, however, that even here we must take vectors into consideration and we would suggest, therefore, the importance of further investigation of the possibility of the conveyance of the disease by blood sucking insects biting patients during the septicemic stage which may cover the period of onset and the period following meningitis as well as the period of actual meningitis. We think such method of conveyance improbable.

Second, as to diagnosis. The irregular form of the fever should always arouse suspicion of a septicemia. Occasionally the recurrent chills and fever, especially at fairly regular intervals, may suggest a malarial infection and enlargement of the spleen may lend color to this assumption. Blood examinations, however, should disprove this. Blood cultures may prove deceptive if certain points are overlooked. Sterile cultures on ordinary media do not exclude bacteremia. Many writers emphasize the importance of using media enriched with blood serum or ascitic fluid. We found, as others did, that blood should be taken early in the rise of the temperature for specimens taken at the height of the peak were much less likely to yield positive results. We believe that this time of taking the cultures (early in the rise) is more important than the type of the media—provided a fairly rich medium is used. We would suggest that the condition is to be regarded as a bacteremia rather than a true septicemia: that is to say the organisms are being sent into the blood stream in showers from a focus (meningeal fold?) and are there only temporarily. They are not growing in the blood stream as in a true septicemia and the blood has not lost its power to fight as in a septicemia. It was very strange that the organisms from the spinal fluid in the original meningitis were agglutinated by the commercial polyvalent diagnostic serum to a dilution beyond 200 whereas in the period of the subsequent meningococcemia the organisms cultivated from the blood were agglutinated only to 1 to 40 by this same polyvalent serum.

On two occasions we tried the agglutinating power of the patient's own blood serum on the organisms cultivated from her blood stream. It did not have any agglutinating power. Also as a matter of interest from the point of view of the immunity process we wish to record here the fact that in the original meningitis we found the spinal fluid was entirely lacking in complement; 1/20 c.c. of guinea pig serum contained more complement than 20 c.c. of the patient's spinal fluid.

Endocarditis lenta will, of course, always be suspected in the presence of irregular fever, joint manifestations and skin eruptions especially when the latter are purpuric. A number of cases of meningococcemia with localization on the heart valves have been reported and this, of course, may cause still further confusion, especially when the meningococcemia is of the pure type without meningeal manifestations. As Dock[4] has pointed out, it is important to avoid this confusion of chronic meningococcemia with bacterial endocarditis because of the great difference in the prognosis as well as of the possibility of finding a specific medication for the meningococcemia. The almost invariable fatal outcome in endocarditis lenta is in a marked contrast with the relatively low mortality of meningococcemia.

The third point to which we direct attention is that of therapy. In studying the cases reported in the literature, especially those in the French literature, we are struck by the fact that the prognosis was remarkably good—no matter what the type of treatment. Sergent[8] considered the chronic meningococcemia the most benign of septicemias. In his observation cures resulted in 90 per cent of the cases. Vesell and Parsky[7] remarked that the mortality in other series has ranged from 10 to 50 per cent. Dock[4] in 1924 has also commented upon this. We are struck also by the apparent futility of all types of treatment. The septicemia continued when any of the following methods were employed: Anti-sera, vaccines, nonspecific protein injections, hypodermic injections of milk, fixation abscesses and intravenous injections of the various forms of arsenic. On the other hand, reports of recovery have been made following the employment of each of these. Again spontaneous recoveries are reported after various methods of treatment had been tried in vain. We are not at all confident that the vaccine treatment of our patient caused her recovery. We rejected the use of anti-serum because of her evident sensitization and our consequent fear of a severe anaphylactic reaction.

Finally, we did not do a lumbar puncture for fear of causing a meningeal localization and a relighting of the meningitis. It has been suggested that the continued septicemia is due to a persistent lurking focus in the meninges from which there is given off from time to time a new infection of the blood stream. This focus is well walled off and none of the meningococci get into the cerebro-spinal fluid, hence the absence of the meningeal symptoms. This theory finds substantiation in the fact that such foci well walled off by adhesions have been found at autopsies of patients dying weeks or months after recovery from meningitis. We felt, therefore, that any sudden disturbance of the equilibrium in the cerebrospinal axis might disturb these adhesions and set the meningococci free in the spinal fluid. Dock,[4] however, says "lumbar puncture in this type of sepsis did not cause immediate meningeal localization in even one of the eighteen cases and subsequent localization in these cases was less frequent than in the whole series." He goes on to remark "However, there is no evidence for the view suggested by Lundy, Thomas, Fleming and MacLagan in regard to the acute type of cerebro-spinal fever that lumbar puncture during the stage of sepsis may arrest the disease, alleviate symptoms, or prevent meningeal infection." We would emphasize in this connection that Dock is discussing meningococcic septicemia preceding an attack of meningitis, whereas in our case we are dealing with the meningococcic septicemia following a previous meningitis. He, therefore, did not have to consider, as we did, the local results of the meningitis.

The fourth observation we wish to make is that our patient's case represents an apparently very rare type. As we have already indicated there are in the literature many reports of chronic meningococcic septicemia which have ended in a fulminating meningitis and there are even reports of

recurrent attacks of meningitis. There are a few cases in which the septicemia remained "pure," that is, it was characterized by the absence of a meningitis at any time. We have been able, however, to find but one case, that of de Lavergne and Carrot[10] where the course of the disease was similar to that of our patient's, a chronic menigococcemia following an acute meningitis. Their patient also recovered. They gave 600 c.c. of serum intramuscularly in the course of five days without the slightest effect upon the temperature. They did not use specific vaccine because it had failed in three other cases of meningococcic septicemia, 1 "pure" and 2 preceding meningitis. They injected their patient (intraspinally?) with his own serum but this produced no result The patient finally recovered apparently spontaneously.

SUMMARY.

A case of meningococcic septicemia of an unusual type following acute meningitis is reported. Recovery followed the use of antogenous vaccines but their value is uncertain. Of great importance is the fact that a brother and a sister of the patient had died of meningitis respectively fifteen and six years previously.

BIBLIOGRAPHY.

1. Gwyn, N.: A case of general infection by the diplococcus intracellularis of Wechselbaum. Phil. Med. Jour. 2:1255, 1898.

2. Herrick, W. W.: Extrameningeal meningococcus infection. Arch. Int. Med. 23:409 ,1919.

3. Soloman, H.: Ueber Menengokokkenseptikamie. Berl. klin. Wehnschr. 39:1045, 1902.

4. Dock, Wm.: Intermittent fever of seven months' duration due to meningococcemia (with an analysis of 68 reported cases of meningococcemia.) Jour. Am. Med. Assn. 83:31. 1924.

5. Marlow, F. W., Jr.: Meningococcemia: report of case with recovery. Jour. Am. Med. Assn. 92:619, 1929.

6. Graves, W. R., Dulaney, A. D., & Michelson, I. D.: Chronic meningococcemia: report of a case. Jour. Am. Med. Assn. 92:1923, 1929.

7. Vesell, Harry, & Barsky, Joseph: Chronic meningococcus septicemia. Am. J. M. Sc. 179:589, 1930.

8. Moleen, G. A. & Seecof, D. P.: Meningococcic septicemia. Colorado Med. 26:192, 1921.

9. Morgan, H. J.: Chronic meningococcus septicemia. Bull. Johns Hopkins Hosp. 32:366, 1921.

10. de Lavergne & Carrot, E.: A propos d'un cas de septicemie meningococcique Bull. et. mem. d. Soc. Med. d. Hop. d. Paris 50:1713, 1926.

DISCUSSION.

Dr. Randolph Lyons (New Orleans): I have been extremely interested in this excellent report of Dr. Lemann's because at Touro we all knew of this case. We were especially interested in the outcome and in the patient.

I have never come across this condition of meningococcemia following meningitis; have never seen a case of pure meningo-coccemia. If I did I failed to recognize the case.

I think one of the points Dr. Lemann brought out is of extraordinary interest—that is the family history. This is a family history in which three patients developed meningitis, possibly one of them almost the same type as Dr. Lemann has described. May we not have here a family susceptible to meningococcal infection as some families are susceptible to typhoid? I might mention my own family in which the father and four children all had typhoid at different times. Another explanation is the possibility of one of the members of the family being a carrier. It has been recently shown that most individuals (up to 90 per cent) are infected by contact with your so-called healthy carriers. These carriers probably become infected by overcrowding with others who have had menigitis, as, for instance, in barracks. We divide carriers primarily into those who have the infection in the naso-pharynx without showing any evidence of disease, and secondarily, into those who harbor the organism following an attack of meningitis. A primary carrier may harbor the organism as long as six weeks; in 10 per cent it may be longer; in 2 per cent nobody knows how much longer. It may be possible for some member of that family to harbor infection for years, but this is purely conjectural.

I have been interested in the question of diagnosis. In the case of Dr. Lemann has described the diagnosis was not quite so difficult because the patient had had an attack of cerebrospinal meningitis. In the pure type, diagnosis might be more difficult. I believe if it had been my case I might have been tempted to get a small amount of spinal fluid as the danger of lighting up the meningitis would have been almost negligible. On the other hand, examination of the fluid might not have thrown any light on the diagnosis.

As far as treatment is concerned, I have never had the opportunity to treat a case and am not in a position to make a statement. Most of them have been given the benefit of anti-serum treatment without any striking effect. When we consider the history of the disease, its chronicity, that a great deal may get well anyway, for any method to be considered specific it would have to give prompt relief.

Dr. J. Birney Guthrie (New Orleans): It is a long time since I have heard as complete a case report as Dr. Lemann gave. If there is any investigation Dr. Lemann should have done and did not, I don't know what it is.

It recalls to me two cases of meningococcic endocarditis I saw at Camp Beauregard during an epidemic there. Both were compiications of meningococcic meningitis. Of these two cases, one died, one was discharged cured. Both cases were rather prolonged in course, going into weeks—even into months. They were treated with meningococcic serum intravenously and by intraspinal route. At that time Herrick insisted on the necessity of treating these cases by the intravenous method. We felt that the focus of the disease had been transferred in both cases. We had no hesitation in making punctures and the findings were negative while the same performance as in this case was going on. There is not one symptom that Dr. Lemann mentioned in the course of this disease: sepsis, leukocytosis, bacteremia, that was lacking in the two cases I saw. Our case which got well cleared up under intravenous injections.

Just a word as to heredity. The relationship of this is an extremely fascinating question. Dr. George S. Draper of the Rockefeller Foundation was at Beauregard with us. He was working along the particular type of individual that was susceptible to meningitis and thought that setting of the ear set below the level of the eye and the meeting of the eyebrow over the nose were more or less indicative of susceptibility. A very interesting observation was made by the undertaker. Every case that died was carefully embalmed; the undertaker heard these discussions and he chimed in with the statement that he found it remarkable that the fauces of so many of the cases that died of meningitis took much more cotton to stuff than those that died of other causes.

It was unfortunate that the poor woman whose history we have heard had an allergy that prevented her having sufficient serum. I cannot think of any condition except terriffic allergy that would contraindicate meingococcic serum in a case of known acute hemingococcic endocarditis. Even under these circumstances, desensitization is possible, and the reactions combatted with epinephine.

The case history suggests acute ulcerative meningococcic menigitis.

Dr. C. W. Duval (New Orleans): Dr. Lemann's case of meningococcemia is of unusual occurrence; in fact, almost unique. Whether meningococcal meningitis is primarily a septicemia, the meninges becoming secondarily involved, has long been a mooted question among bacteriologists. I have always held that the primary invasion was of the meninges. In my experience only a small percentage of cases show septicemia, and in these the blood invasion has occurred late.

In this case of Dr. Lemann's I am inclined to believe with Dr. Guthrie that after the meningitis cleared up there remained a local immunity of the meninges which would explain reinfection of the brain and cord coverings did not occur during the period in which the meningococcus apparently flourished in the blood stream. The intermittent occurrence of meningococci in the blood undoubtedly was the result of a shower from some focus of infection outside of the central nervous system. The most likely places for such a focus would be the mucous membrane of the nasal pharynx, the sinuses and endocardium, including the valves of the heart. Probably at the time of the meningeal clear up in this particular case the focus was endocarditis, from which source the blood received intermittent showers of meningococci.

Dr. I. I. Lemann (closing): It was necessary to omit some of the paper. One of the things Dr. Teasley alluded to was that, in the original illness, acute meningitis, he found a difference in the agglutinating power of the commercial serums. We tried first one, then another, and then a third, which last had greatest power and the greatest improvement followed the use of this. That does not necessarily means that the third serum was most potent.

As to Dr. Guthrie's suggestion, reinforced by Dr. Duval, in regard to the involvement of the heart, I would only say that daily examination of the heart failed to reveal anything that would suggest any heart involvement; no murmur, no roughening of sound, nothing that suggested any trouble of the heart, nor was there any tenderness over the sternum so suggestive in cases of endocarditis.

As was suggested by Dr. Duval, that clinicians cannot always determine in endocarditis, the valve involved, as witness the case of Dr. Whipperman. I can only say that we heard first a murmur, then a somewhat louder murmur, then a very loud, rasping murmur that grew louder and more distinct up to the time of his death. Our only mistake in diagnosis was as to the location.

INFLUENZAL MENINGITIS IS AMENABLE TO TREATMENT.*

CHARLES JAMES BLOOM, M. D.,†

NEW ORLEANS.

INTRODUCTION.

The pestilence mentioned by Homer (Iliad, Book I) and the sweating sickness of the fifteenth century were, perhaps, epidemics of influenza.

In 1892, Pfeiffer, Canon[21] and Kitasato,[69] independently, isolated the organism that now bears the name "bacillus influenzae." The pathogenecity of the organism is still doubted by the bacteriologists. Contrary to original observations of the discoverers, the bacillus can be found in inter-epidemic periods without an associated influenza. In addition, animal experimentations have been very unsatisfactory and other than the deduction made by Wollstein[123] (1911) relative to influenzal meningitis, no definite specificity can be proven regarding this organism.

Slawyk[107] (1899) definitely established influenzal meningitis as a clinical entity, the bacteriological work being supervised by Pfeiffer himself. Hogerstedt[56] (1895), Pfuhl[94] (1897), Haedke[51] (1897) and Testivini[114] (1897) recorded cases with questionable bacteriological findings.

The first case recorded in the United States was by Adams[3] in 1907. Though the disease is uncommon, a fairly large number of cases have been reported, and with more careful bacteriological study of the cerebro-spinal fluid in the future, statistics will reveal a much large number of cases than heretofore diagnosed.

Neal[89] (1921), analyzing 692 cases of purulent meningitis occurring during a period of ten years, 1910-1920, found only 5 per cent of the influenzal type, and Dunn[38] (1911), in a series of 82 cases, recorded four.

INCIDENCE.

According to Dunn[38] (1911) and Neal[89] (1921), meningitis produced by B. influenzae ranks fourth in order among the types of purulent meningitis, as follows: 1. Meningococcic; 2. pneumococcic; 3 streptococcic; 4. influenzal.

The following Hospital statistics are here recorded:

The Children's Hospital, Boston, Mass.

1928 and 1929

Medical Diseases treated in:		1928	1929
a. Outpatient Department			
	Meningitis, meningococcus	18	14
b. Wards			
	Meningitis: meningococcus	12	10
	tuberculosis	7	12
	pneumococcus	3	3
	influenza bacillus	2	3
	streptococcus	—	6
	staphlycoccus	—	1

Touro Infirmary, New Orleans, La.

1924-1930

***

Meningitis:	pneumococcus	21
	meningococcus	12
	influenza bacillus	12
	streptococcus and staphylococcus	11
	colon bacillus	2
	organism undetermined	6

*** This includes all cases—adults and children.

No classification of types could be obtained from published records of Charity Hospital, New Orleans, La., nor from the City Board of Health of the Parish of Orleans.

Levin[78] (1920) studied the complications of 816 cases of influenza in children at the

*Read before the Orleans Parish Medical Society, June 9, 1930.

†From the Department of Pediatrics, Graduate School of Medicine, Tulane University.

Cook County Hospital in Chicago during the epidemic and only 5 cases of meningitis were found, and none of these from bacillus influenza.

SURVEY OF LITERATURE.

Rivers[98] (1922) made an almost complete survey of the literature up to 1922.

Table I of this paper adds to Rivers seventeen recoveries, six that have since been recorded in the literature and one from the writer's series of personal cases.

Table II comprises Rivers' list of authentic cases of influenzal meningitis (deaths), with the exception of numbers 10, 43, 62, 65, 67, 73, 77, 79, which are reported by Schnyder and Urech[103] (1925) as "cases where the pure culture of liquor of the meninges have shown Pfeiffer's bacillus."

Table III is the result of the author's survey of the literature of the United States—and some foreign references—from 1921 through March, 1930. When obtainable, original articles were reviewed; where this was not possible it is so indicated.

Table IV records cases of purulent and tuberculous meingitis, from the writer's private practice, from 1916 up to April, 1930.

Table I—Authentic Cases of Influenzal Meningitis. (Recovered).

No.	Name of Author	Year	Country	Age	Sex	Treatment
1	Langer[76]	1901	Germany	9 yrs.	M	L. P.
2	Mya[87]	1903	Italy	9 mos.	—	L. P.
3	Thomesco and Grascoski[115]	1905	Roumania	7 yrs.	—	L. P.
4	Cohoe[28]	1909	United States	33 yrs.	M	L. P.
5	Batten[8]	1910	Great Britain	14 mos.	F	L. P.; Anti-Menin. Serums Urotropin
6	Sayce[101]	1911	Australia	4 yrs.	M	L. P.
7	Klinger[71]	1912	Germany	26 yrs.	—	*Intravenous injections electragol daily for one week.
8	Spillmann and Benech[108]	1913	France	Adv. age	M	L. P.; Anti-Menin. Serum.
9	Hill and Packard[54]	1915	Panama	4 mos.	M	L. P.
10	Packard[92]	1916	United States	11 yrs.	F	L. P.; Anti-Influenzal Serum.
11	Torrey[117]	1916	United States	11 yrs.	F	L. P.; Anti-Menin. Serum; Anti-Influenzal Serum.
12	Johnson[61]	1919	United States	3 yrs.	M	L. P.; Convalescent Serum Intraspinally and Subcutaneously.
13	Stone[111]	1920	United States	15 yrs.	F	L. P.
14	Litchfield[80]	1920	Australia	23 mos.	F	L. P.; Autogenous Vaccine.
15	Christiansen and Kristensen[24]	1921	Denmark	2 yrs.	M	L. P.; Anti-Menin. Serum.
16	Neal[89]	1921	United States	2½ yrs.	F	L. P. and Vaccine Intraspinally; Anti-Influenzal Serum.
17	Rivers[98]	1922	United States	13 mos.	F	L.P.
18	Hadfield[50]	1922	Great Britain	14 yrs.	M	L. P.
19	Jordan[63]	1924	United States	16 yrs.	M	L. P.; Anti-Menin. Serum; Drain. and washing spinal canal Physiol. salt solution.
20	Kristensen and Christensen[74]	1924	Denmark	8 yrs.	M	L. P.; Anti-Pneu. Serum.
21	Schnyder and Urech[103]	1925	Germany	4 yrs.	M	L. P.; Menin. Serum.
22	Turner[119]	1926	United States	6½ yrs.	M	L. P.; Anti-Menin. Serum..
23	Signorelli[102-a]	1929	United States	7 yrs.	F	L. P.
24	Bloom	1930	United States	10 mos.	F	L. P.; Convalescent Serum.

*Original article not obtainable; treatment cited by Torrey[117](1916).
L. P.—Lumbar Puncture.

TABLE II.

No.	Authority	Year	No. of Cases	No.	Authority	Year	No. of Cases
1.	Fraenkel[44]	1898	2	41.	Giese[47]	1913	1
2.	Slawyk[107]	1899	1	42.	Ducrot†[36]	1913	1
3.	Meunier[85]	1900	1	43.	Grasty[48]	1913	1
4.	Trailescu[118]	1901	1	44.	Ross and Moore[99]	1913	1
5.	Dubois[35]	1902	1	45.	Wollstein[125]	1913	3
6.	Ghon[46]	1902	1	46.	Atkinson[7]	1914	1
7.	Cagnetto[20]	1903-4	2	47.	Tebbutt[112]	1914	1
8.	Costinesco[29]	1903	3	48.	Hubschmann[58]	1914	1
9.	Mya[87, 52]	1903	3	49.	Nyberg[91]	1915	2
10.	Hecht[67]	1903	1	50.	Ely and Weingart[40]	1915	1
11.	Jundell†-[1]	1904	2	51.	Imperatori[60]	1915	1
12.	Bertini[11]	1904	1	52.	Cleland and Ferguson[25]	1915	2
13.	Cattaneo[28]	1905	1	53.	Brown[16]	1915	2
14.	Simon‡[105]	1905	1	54.	Boland[13]	1915	1
15.	Dudgeon and Adams[37]	1907	1	55.	Hill and Packard[54]	1915	4
16.	Sprigg[109]	1907	1	56.	Aaser[1]	1916	1
17.	Douglas[34]	1907	1	57.	Moody†[86]	1916	2
18.	Saathoff[100]	1907	1	58.	Williams[121]	1916	1
19.	Adams[3]	1907	1	59.	Torrey[117]	1916	1
20.	Bentz and Frye[10]	1908	1	60.	Bhat[12]	1917	1
21.	McDonald†[84]	1908	1	61.	Tobler[116]	1917	1
22.	Hymanson[59]	1909	2	62.	Lorey[81]	1917	1
23.	Cohen[27]	1909	3	63.	Lacy[75]	1918	2
24.	Davis†[32]	1909	2	64.	Hills[55]	1918	1
25.	Simon and Aine[106]	1910	5	65.	Oeller[92]	1918	1
26.	Simon[104]	1910	2	66.	Albert§ & Kelman[5]	1919	1
27.	Batten[8]	1910	4	67.	Hotz[57]	1919	2
28.	Ager and Avery[4]	1910	1	68.	Dunn[39]	1920	10
29.	Ritchie†[97]	1910	3	69.	Christiansen & Kristensen[24]	1921	2
30.	Sayce[101]	1911	6	70.	Finkelstein[43]	1921	1
31.	Clemens and Gould[26]	1911	1	71.	Abt[2]	1921	1
32.	Dunn[38]	1911	4	72.	Koplik§	1921	9
33.	Rhea[96]	1911	2	73.	Boyowitsch[14]	1921	3
34.	Prasek and Zattelli[95]	1911	1	74.	Neal[89]	1921	31
35.	Wollstein[124]	1911	7	75.	Neal§	1921	3
36.	Brem and Zeiler[15]	1911	2	76.	Davis§	1921	2
37.	Davis[32]	1911	5	77.	Kotz[73]	1921	2
38.	Jundell[68]	1912	1	78.	Ill§	1922	1
39.	Klinger[71]	1912	3	79.	Bender[9]	1922	2
40.	Henry[53]	1912-13	7	80.	Rivers[98]	1922	22
				81.	Erb[42]	1921	9

†Reported also in 1902 and counted twice by Simon in 1910.

‡Probable case.

§Personal communication—11 reported but one included under Wollstein, 1913.

Table III—Authentic Cases of Influenzal Meningitis.

No.	Authority	Year	No. of Cases	Country	Age	Sex	Treatment
82	Jordan and Smith[65]	1921	1	U. S.	14 mos.	F	L. P.; Anti-Menin. Serum; Spinal Wash and Drain. Subarachnoid Space.
83	de Leon[32]	1921	1	P. I.	7 mos.	M	L. P.
84	Manson[82]	1922	1	G. B.	7 mos.	F	L. P.
85	Templeton, King and McKean[113]	1922	1	U. S.	17 mos.	M	L. P.; Anti-Menin. Serum; Menin. Serum.
86	Butler[13]	1923	1	G. B.	4½ mos.	F	L. P.
87	*Engering[41]	1923	4	Germ.			
88	*Lisbonne and Leenhardt[79]	1923	1	France	infant		
89	Worster-Drought[127]	1923	1	G. B.	62 yrs.	M	L. P.; Pfeif. Bac. Vac.
90	Nabarro and Stallman[88]	1924	1	G. B.	1 yr.	F	Autogenous vaccine.
91	*Klinke[73]	1924	1	Germ.			
92	Buol[17]	1924	1	U. S.	3½ yrs.		L. P.; Anti-Menin. Serum; Wash. Spinal canal physiological salt.
93	*Gerbasi[46]	1924	1	Italy	infant		L. P.
94	*Schmidt and Weinberg[102]	1924	1	Germ.	child		L. P.
95	Greenthal and Kelly[49]	1924	5	U. S.	1 yr.	F	L. P.
					21 mos.	M	L. P.
					7½ mos.	M	L. P.
					11 mos.	F	L. P.
					16 mos.	M	L. P.
96	*Leuchtenberger[77]	1924	3	Germ.			
97	Schnyder and Urech[105]	1925	6	Germ.	2 yrs.	F	L. P.; Menin. Serum
					9 mos.	M	
					2½ yrs.	F	
					11 mos.	M	L. P.; Streptoc. Serum.
					20 mos.	M	L. P.; Menin. Serum; Pfeiffer Multivalent Serum.
					22 mos.	M	L. P. (Immunogen) Infl. Serum.
98	Kincaid[70]	1926	1	Australia	4 mos.		L. P.; Anti-Bact.; Anti-pneumoc. Serum.
99	Marcus-Crane[83]	1928	1	U. S.	16 mos.	F	L. P.
100	Nedelmann[90]	1929	6	Germ.	†		
					1¾ yrs.	F	L. P.
					8½ mos.	F	L. P.
					1½ yrs.	F	**
					13 mos.	M	L. P.
					1¼ yrs.	F	L. P.
					1 yr.	M	Pleuropuncture; L. P.
101	Strange‡	1930	6	U. S.	3½ yrs.	M	L. P.
					51 yrs.	M	L. P.
					1½ yrs.	F	L. P.
					8 mos.	M	L. P.
					7½ mos.	M	L. P.
					2 yrs.	M	L. P.
102	Bloom	1930	9	U. S.	2 mos.	M	L. P.
					22 mos.	F	L. P.
					5 mos.	M	L. P.
					4 yrs.	M	L. P.
					8 mos.	M	L. P.
					2 yrs.	M	L. P.
					4 yrs.	F	L. P.
					4 yrs.	F	L. P.
					32 mos.	F	L. P.

*Original reference not obtainable.

**Child died before admission; diagnosis made at autopsy and the bacteriologic examinations on the dissecting table.

†Also published by V. Gavel (Dissertation, Jena, 1927).

‡Personal communication to author; Dr. Jack Strange, New Orleans, La.—all cultures positive.

Table IV—Author's Cases of Purulent and Tuberculous Meningitis*
1916-1930†

	No. Cases	Died	Recovered	Year	Season—Month	Sex	Age
INFLUENZAL MENINGITIS Maximum Blood Count Total 31,500; N. 89%; S. 8%; L. 3% (Age 4 yrs.) Minimum Blood Count Total 6,500; N. 55%; S. 43%; L. 2% (Age 2¾ yrs.) Spinal Fluid Cell Count Maximum 1000 Minimum 254 (Predominance of polymorphonuclears.)	10	9	1	1916–1 case 1923–2 cases 1925–1 case 1926–1 case 1927–1 case 1929–2 cases 1930–2 cases	Winter 6 cases Spring 2 cases Autumn 2 cases Jan. 1 case Feb. 3 cases Apr. 2 cases Aug. 1 case Nov. 1 case Dec. 2 cases	M–5 cases F–5 cases	Under 1 yr.–4 cases 1 to 2 yrs.–1case 2 to 3 yrs.–2 cases 4 to 5 yrs.–3 cases
TUBERCULOUS MENINGITIS	6	6	0	1917–1 case 1918–1 case 1922–2 cases 1925–1 case 1926–1 case	Spring 2 cases Summer 3 cases Winter 1 case	M–5 cases F–1 case	Under 1 yr.–4 cases 2 to 4 yrs.–2 cases
PNEUMOCOCCAL MENINGITIS	5	5	0	1923–1 case 1925–1 case 1927–1 case 1929–1 case 1930–1 case	Winter 3 cases Summer 2 cases	M–4 cases F–1 case	Under 1 yr.–4 cases 1 to 2 yrs.–1 case
MENINGOCOCCAL MENINGITIS	4	2	2	1923–1 case 1926–1 case 1927–1 case 1929–1 case	Winter 3 cases Summer 1 case	M–2 cases F–2 cases	Under 1 yr.–1 case 3 to 5 yrs.–2 cases Over 10 yrs.–1 case
STREPTOCOCCAL MENINGITIS	2	2	0	1917–1 case 1928–1 case	Autumn 1 case Winter 1 case	M–2 cases	0 to 2 yrs.–2 cases

*Private practice. †Up to April, 1930.

BACTERIOLOGY AND PATHOGENESIS.

The bacillus influenzae is one of the smallest rod-shaped organisms isolated up to the present time. It is nonmotile, aerobic, gram negative, pleomorphic, and is grown on media containing blood. On the ordinary nutrient media no growth is obtained, but in spinal fluid its varied type is more marked and its growth is noted both extracellular and intracellular. Human blood was used in my case to be reported, though Wollstein utilized rabbits' blood alone in her experiments. The organism stains rather readily, more intense at the poles. At the end of 24 hours, translucent elevations can be seen on media. After 72 hours, incubation and no growth is noted, it is pronounced negative.

Jacobson (cited by Cohoe[28]) found that an increase in virulence resulted when the influenzal bacilli were injected in animals along with the cultured streptococcus. This may account for the fact that contamination of cultures seemed to accentuate the growth of the colonies.

The spinal fluid serves as the means of determining the diagnosis. A gram negative stain of a smear made from the centrifugalized sediment should suggest

the possibility of an influenzal infection; meningococcus and colon bacillus must necessarily have to be eliminated as they, too, are gram negative. A part of the fluid is now cultured on blood agar.

Regarding the prognosis, Pfeiffer himself, in a series of many experiments on mice, rats, guinea pigs, rabbits, hogs, cats, dogs and monkeys was unable to produce a true infection. This alone he induced in apes. Pfeiffer has shown the toxin to be intracellular, but Cantanni[22] (1896) demonstrated that this endotoxin had a predilection for the central nervous system, for he found that from two to six mg. of the dead bacteria were sufficient to kill an animal of from 1500 to 2000 grams.

The question of the portal of entry of the organism into the central nervous system has been a topic of much dissenting discussion. From statistical data, respiratory influenza is by far the most common type, therefore, the naso-pharynx is suggested as the site of entry. Fraenkel's[44] (1898) hypothesis in accord with the above statement, however, is not borne out by post-mortem findings as nasal secretions in these cases have been negative for the influenza bacillus.

There are many conceptions regarding the nature of influenzal meningitis: (1) Secondary to respiratory infection; (2) part of a general infection, and (3) a primary meningitis—all of these theories having their respective champions.

In substantiation of (1) the primary foci of infection has been attributable (a) frontal sinus infection; (b) otitis media; (c) influenzal pneumonia; (d) acute rhinitis and bronchitis; (e) epiphysitis of the radius; (f) hematoma—in each instance the influenza organisms having been recovered.

(2) This conception has but a few followers. Dudgeon and Adams[37] (1907) and Cohen[27] (1909) accept this premise.

(3) The production of an experimental meiningitis by Wollstein and her conclusions has aided the belief that this clinical entity is a primary meningitis. Additionally, if one considers that in 3400 cases of influenza (Stangl[110] 1919) only 1 per cent of these patients had symptoms suggestive of meningitis of cerebral involvement, and no post-mortem revealed a meningitis, we can realize that the incidence in meningitis bears no relation whatsoever to the occurrence of epidemics. Jordan[62] (1920), Neal[89] (1921), and myself, at this time, from statistics herein given, find no great increase in influenzal meningitis during and after the pandemic of influenza in 1918 and 1919.

SEASONAL INCIDENCE.

Rivers[98] (1922) found most of his cases occurred in October, November, December and January, and Neal[89] (1921) made a similar observation — October, November and December; in this series, December, January, February and April. The curve of influenzal meningitis in New Orleans seemingly follows the acute respiratory diseases, differing somewhat from that heretofore published by others.

BIOLOGIC AND SEROLOGIC REACTION OF DIFFERENT INFLUENZA BACILLI.

It is an accepted fact that in most cases of influenzal meningitis there has been a close relationship culturally and serologically between the strains producing the disease. This has not been proved regarding influenzal infections in any other part of the human anatomy. Unfortunately, time precludes my discussing this subject in detail. Jordan and Sharp[66] (1922) gave valuable conclusions in their summary:

"The particular strain of Pfeiffer bacillus used to produce an immune serum is usually agglutinated by the homologous serum more rapidly, more constantly and in higher dilution than any heterologous strain.

"Absorption tests throw little if any more light on the true biologic relationship

of the different strains in this group than does direct agglutination.

"As a rule, each strain of Pfeiffer bacillus possesses a serologic individuality.

"Occasionally strains from independent sources exhibit a serologic identity. This is not common.

"As many as three serologic races may be present at the same time in the throat of one patient.

"There is no correlation between indol-producing powers and agglutinative affinities, except possibly in the strains isolated from meningitis.

"The lack of any definite serologic grouping among the strains of Pfeiffer bacilli is an indication that a distinct invasive type, or types, has not become fixed, and is an argument against regarding any member of this group as the primary causal agent in epidemic influenza. Possibly a race of 'influenza meningitis' bacilli is in process of evolution."

AGE AND SEX.

Age.

Rivers[98] (1922), in a series of 193 cases in which age was stated, found 152 under two years and 41 two years and older.

Of the 24 recoveries recorded in Table I the ages were as follows:

To 1 year	3 cases
1-2 years	4 "
2-5 years	4 "
5-7 years	3 "
7-11 years	4 "
11-16 years	3 "
16† years	3 "
	24 cases

Of the 53 deaths comprising Table III the ages were:

To 2 years	35 cases
2-5 years	7 "
"Child"	1 case
50† years	2 cases
Age not given	8 "
	53 cases

Sex.

Cohoe[28] (1909) says "males have been attacked more frequently than females in the cases reported, in which the sex was stated, in the proportion of 16 to 6." In Table I of this paper, recording 24 recoveries, we find 12 males, 9 females, and 3 cases in which sex was not stated; in Table III listing 53 deaths, there were 22 males, 18 females, and 13 cases in which the sex is not known.

MORTALITY.

Of 302 cases recorded in this paper, there were 24 recoveries, a mortality of 92.05 per cent. Rivers,[98] in his series of 220 cases, had a mortality of 92.25 per cent. The death rate for recorded cases, therefore, appears to be about 92 per cent. However, it is my belief that this figure would be nearer 98 per cent if all the deaths were reported; the recoveries usually are.

POSSIBILITY OF SPREAD BY CARRIERS.

Rivers[98] (1922) states as follows: "So few cases of influenzal meningitis occur that it is impossible to show that the disease is spread by contact. There are facts, however, that might make one think that it is spread by carriers. Many observers report several cases occurring within a short time of each other. Hill and Packard[54] (1915) saw four cases in six months. Henry[53] (1912) observed at least five cases in 1910. In Dr. Howland's clinic seven cases were seen in one year. Simon and Aine[106] (1910), in reporting five cases, speak of them as a little epidemic of influenzal meningitis. Davis[31] (1909) reported that twins 5 days old became ill at the same time, one died on the ninth day, the other of the twelfth. At the necropsy the first one was found to have had an influenzal meningitis. No lumbar puncture or necropsy was made in the second case. Although there may be some doubt about the second child having a meningitis, yet it is reasonable to suppose that it did as it became ill at the same time and ran a course similar to the other. Davis[32] (1911)

also reported two cases of influenzal meningitis that occurred in a small institution of about fourteen inmates. The second child became ill two weeks after the death of the first. While not convincing, the evidence is at least suggestive that influenzal meningitis is a carrier-borne disease."

TREATMENT.

Pertinent to treatment, there are numerous measures recommended which deserve serious and continued consideration.

(1) The anti-influenzal serum
   a. Wollstein (1911)[123]
   b. Convalescent serum
(2) Hexamethylenamin
(3) Repeated lumbar puncture with or without the use of normal saline washing
(4) Vaccine
   a. Intravenous
   b. Intraspinal
   c. Combination
(5) Combination of 1-2-3-4

(1-a) Wollstein[123] (1911) produced experimental meningitis in monkeys by injecting virulent influenza bacilli intraspinally. She subsequently found that all of the monkeys developed positive blood cultures as well as positive nasal secretions. When the meningitis was cured by the use of her serum the nasal secretion and blood cultures became negative. She obtained an anti-serum in goats which cured, experimentally, meningitis in monkeys. Unfortunately, results in humans have been unsuccessful in all but a few cases, and at this time the serum is not available. The specific serum was made by immunizing goats for a period of 18 months with known strains of influenzae bacilli. This in turn possessed a large amount of agglutinins and opsonins. The cases of experimental meningitis were cured by immediately following the injection of influenza bacilli into the spinal canal, with an injection of the antiserum; this was performed at intervals of one hour in some cases; in others after 9 hours, and a third group after 24 hours. In most cases cures seemed instantaneous; the monkeys developing the usual signs and then convalescing in place of dying. The incubation period was from five to eighteen hours, and usually when the serum was omitted, the animals died within a period of one-half to four days. Dunn[38] (1911) reports having used it in eleven cases without recovery, but inadequate doses may have been responsible for his conclusion.

(1-b) Described in author's case, also Packard[93] (1916); Torrey[117] (1916); Johnson[61] (1919); Neal[89] (1921). (These cases are included in Table I—Recoveries).

(2) Crowe[30] (1909) observes "that after doses of urotropin, within therapeutic limits, a sufficient amount of the drug appears in the cerebrospinal fluid to exercise a decided inhibitory effect on the growth of the organism and will markedly defer, and in some cases prevent, the onset of fatal meningitis." Recommended by Brem and Zeiler[15] (1911) and Batten[8] (1910), who, incidentally reports a case, with recovery, following its use.

(3) Torrey[117] (1916) remarks "Drainage of the subarachnoid space by means of lumbar puncture has been practiced extensively since it was first proposed by Quincke. Many writers make the assertion that in meningitis this procedure may relieve pressure symptoms, but can have no curative effect. In view of the reports of severe cases of meningitis, even of the pneumococcic and tuberculous varieties, showing recovery following repeated lumbar puncture, these statements seem unwarranted. There is an immediate and very striking temporary good effect, probably due directly to the relief of excessive intracranial tension and improved blood supply in the tissues of the brain and cord. There is also removal of the toxins which would otherwise be absorbed into the circulation. Improvement in the circulatory conditions following decreased pressure may have an

effect in combating local infection. It is believed that under normal conditions the cells of the choroid plexus, in secreting "the cerebro-spinal fluid, act as an effective barrier to most toxins and antibodies. It has not, as far as I know, been demonstrated that antibodies do not pass into the spinal fluid in cases of meningitis where there is an active inflammation of the membranes, and leukocytes pour into the fluid in enormous numbers, and the conditions are far from normal. The increased phagocytosis and decrease in organisms which has been demonstrated following lumbar puncture suggest that with the resulting lowered pressure, and increased outpouring of fluid into the cerebrospinal space, protective substances may pass from the circulation into the spinal fluid."

"A further benefit probably derived from frequent spinal drainage is protection in part against the development of that much feared complication of meningitis—internal hydrocephalus—as the rapid flow of fluid from the ventricles tends to keep the foramen of Magendie clear of exudate which might cause obstruction at this point. . . "

(3) Buol[17] (1904) suggests washing the spinal canal with physiological salt solution every 8 hours after the administration of an intraspinal injection of a serum.

Jordan[63] (1924), reporting a case having recovered, used in his routine, daily washing of the spinal canal with physiological salt solution. This he did for five successive days.

(4) Treatment with autogenous vaccine was recorded by Nabarro and Stallman[88] (1924) and by Neal[89] (1921). Neal, in summarizing her report, says: "I wish to make it clear that I do not assert that intraspinal vaccine treatment cured the case of influenzal meningitis that I have described nor that the intravenous administration of the vaccine cured the case of blocked epidemic meningitis to which I referred. However, recovery did follow the use of vaccine in these cases."

Abt and Tumpeer[2] (1921) conclude, by stating "the most rational form of treatment involved to date consists in getting the normal or convalescent serum and subsequent aspirations of spinal fluid." The serum necessarily contains the antibodies which should act as an aid to increase phagocytosis while the lumbar puncture relieves the pressure and aspirates the fluid.

CASE REPORT.

Gladys S., white female infant, 10 months of age, was admitted to the Touro Infirmary December 17, 1929, having been referred by Dr. J. I. Woodward of Picayune, Mississippi. She was stricken with high fever which had continued for six days prior to her admittance.

But little knowledge was gained by physical examination. Her temperature was 105°F., rectum, restlessness was extreme, respiration rapid and shallow. There was some redness of the throat, and the right drum showed some pathology. With continued observation, and with no improvement following a paracentesis, the picture shifts to an increasing state of lethargy.

Let it be clearly understood that up to this time there was no suggestion of meningitis. On the sixth day in hospital, there was bulging of the anterior fontanelle, rigidity of the neck, with positive neurological findings. A spinal puncture was performed and a hazy fluid, under increased pressure, with smear and culture positive for B. influenzae was found. The cell count was 1,000 and the globulin +++.

At this juncture the parents were told that but little hope of recovery could be given to them, and permission was granted for the use of convalescent serum. Accordingly, a consultation was held with Dr. Vesey Johnson*, and a plan of treatment was agreed upon.

*Pathological Intern, Staff, Touro Infirmary, New Orleans.

Daily spinal punctures were done and spinal fluid removed until the baby became restless; 20 to 30 c.c. were drained each day. Immediately following this procedure, human serum was injected intraspinally, the amount being a few cu. cm. less than the spinal fluid removed. The father was the donor; he had had influenza a few months before. Sufficient blood was collected in 50 c.c. sterile test tubes so that each tube yielded about 20 c.c. of serum; five tubes were filled each time. The blood was allowed to clot, and the clot to retract, tubes remaining in the refrigerator for about 12 hours. The tubes were centrifugalized, the serum removed, and that from each large test

tube stored in smaller tubes until used—thus 20 c.c. of serum was placed in each tube. It is interesting to note that the serum given the baby would not agglutinate the influenza bacilli cultured from the spinal fluid. As a matter of record, the mother had recovered from grippe just before the baby became ill.

Twenty-nine days after admission the baby was discharged, with apparently no aftermath. I saw her last on the 14th of March, 1930 and she seemed normal in every respect. I expect to show her in person tonight.

Chart 1—Recapitulation—Gladys S.—Recovered.

Cultures	Punctures	Date
		Dec. 17, 1929
		18, 1929
		19, 1929
		20, 1929
		21, 1929
		22, 1929
Positive	Spinal Puncture	23, 1929
"	13 c.c. serum	24, 1929
Negative	20 c.c serum	25, 1929
"	18 c.c. serum	26, 1929
Positive	15 c.c. serum	27, 1929
"	10 c.c. serum	28, 1929
Negative	Spinal Puncture	29, 1929
"	15 c.c. serum	30, 1929
Positive	Spinal Puncture	31, 1929
"	Spinal Puncture	Jan. 1, 1930
Negative	Spinal Puncture	2, 1930
"	10 c.c. serum	3, 1930
"	10 c.c. serum	4, 1930
Positive	Spinal Puncture	5, 1930
"	15 c.c. serum	6, 1930
"	15 c.c. serum	7, 1930
"	10 c.c. serum	8, 1930
No report	No Puncture	9, 1930
Negative	10 c.c. serum	10, 1930
No report	No Puncture	11, 1930
Negative	Spinal Puncture	12, 1930
No report	No Puncture	13, 1930
Negative	Spinal Puncture	14, 1930
Discharged.....	Well	15, 1930

Consultation—Ear:

Dec. 20, 1929: There is some redness of the right drum but not enough to warrant tympanotomy at this time.

Dec. 21, 1929: Right drum opened.

Jos. D. Martin, M. D.

Roentgen-ray Consultation:

Dec. 24, 1929: Both diaphragms, heart and aorta are well within normal limits. The pulmonary fields are quite clear, and from the roentgen standpoint there is no visible evidence of pathology.

W. F. Henderson, M. D.

Clinical Laboratory:

Dec. 31, 1929: Urine: Acid; Straw; NSQ; Sediment: Occasional pus cell.

Dec. 17, 1929: Blood: T.W.C. 17,400, N. 72, S. 22.

Jan. 7, 1930: T.W.C. 6,500, N. 76, S. 21, L. 3; T.R.C. 3,250,000.

SUMMARY.

1. The records of 302 cases have been reviewed, including ten cases observed by the writer.

2. Resume of all literature published in the United States, and some in foreign countries, up to March, 1930, is made.

3. The mortality in 302 cases was 92.05 per cent.

4. Seven additional cases of recovery are added to Rivers[98] (1922) table, including one by the author.

5. Influenzal meningitis is a disease of infancy, as statistics herein prove.

6. The relationship between the seasonal and yearly incidence of influenzal meningitis and of epidemic influenza and pneumonia do not coincide, though this series occurred somewhat later than that reported by others.

CONCLUSIONS.

1. Influenzal meningitis seems to be a primary infection influenced by different strains of bacilli which culturally and serologically are closely related.

2. The report of a case treated with human convalescent blood serum, with recovery and no aftermath, should be an inspiration for further study and continued usage.

BIBLIOGRAPHY.

1. Aaser, E.: Tidsskr. f. d. norske Laegefor.; 36:393, 1916.

2. Abt, I. A. & Tumpeer, I. H.: Influenza meningitis, with report of case. Am. J. Dis. Child.; 21:444, 1921.

3. Adams, S. S.: Arch. Pediat.; 24:721, 1907.

4. Ager, L. C. & Avery, O. T.: Arch. Pediat; 27:284, 1910.

5. Albert, H. &. Kelman, S.: J. Infect.; 25:433, 1919.

6. Anderson, R. A. & Schultz, O. T.: Immunologic study of strains of bacillus Pfeiffer isolated from a case of meningitis. J. Exper. Med.; 33:653, 1921.

7. Atkinson, E.: M. J. Australia; 1:421, 1914.

8. Batten, F. E.: Lancet; 1:1677, 1910.

9. Bender: Zbfl. bact.; Abt. I. Origin. Bd. 87, 1922.

10. Bentz, C. A. & Frye, M. J.: Woman's M. J.; 18:73, 1908.

11. Bertini, E.: Riv. clin. Pediat.; 2:673, Firenze, 1904.

12. Bhat, K. S.: Lancet; 2:384, 1917.

13. Boland, C. V.: Lancet, 2:704, 1915.

14. Boyowitsch: Inaug. Dis. Zurich, 1921.

15. Brem, W. V. & Zeller, A. H.: Am. J. Dis. Child.; 1:417, 1911.

16. Brown, A.: Canad. M. A. J.; 5:1077, 1915.

17. Buol, Robt. S.: Influtnzal meningitis; Rhode Island M. J.; 7:15, 1924.

18. Butler, E. N.: Meningitis due to Pfeiffer's bacillus; Brit. M. J., 1:719, 1923.

19. Caccia: La Pediatria; 1903, 172.

20. Cagnetto, G.: Atti d. Reale Inst. Veneto, 63:Pt. 2, 397, 1903.

21. Canon: Cited Jordan's Textbook Bacteriology.

22. Cantani: 1896; Stschr. f. hyg.; xxiii.

23. Cattaneo, C.: Rend. d. Assn. med. chir. di Parma; 6:115, 1905. Abstr.: Centralbl. f. Bakteriol. (Ref) 39:455, 1907.

24. Christiansen, M. & Kristensen, M.: Meningitis from influenza bacillus; four cases. Acta med. Scandinav., 55:298, June 1921 (In French); Ugesk. f. Laeger, 83:551, 1921.

25. Cleland, J. B. & Ferguson, E. W.: M. J. Australia; 1:308, 1915.

26. Clemens, J. R. & Gould, C. W.: Arch. Pediat.; 28:210, 1911.

27. Cohen: Ann. de l'Inst. Pasteur; 23:273, 1909.

28. Cohoe, B. A.: Influenzal meningitis; Am. J. M. Sc. 137:74, 1909.

29. Costinesco: Congress Internat. d. Med.; 1903, Pediat. Sec. p. 967.

30. Crowe: Johns Hopkins Hosp. Bull.; 20:102, 1909.

31. Davis, D. J.: Arch. Int. Med.; 4:323, 1909.

32. Davis, D. J.: Am. J. Dis. Child.; 1:95, 1911.

33. deLeon, W.: J. Philippine Isl. Med. Assn.; 1:195, 1921.

34. Douglas, J.: Lancet; 1:86, 1907.

35. Dubois: These de Paris; Nov. 27, 1902, No. 49. Cited by Adams, Arch. Pediat.; 24:721, 1907.

36. Ducrot: Rev. Med. de la Franche-comite; 21:195, 1913.

37. Dudgeon, L. S. & Adams, J. E.: Lancet; 2:684, 1907.

38. Dunn, C. H.: Am. J. Dis. Child.; 1:95, 1911.

39. Dunn, C. H.: Pediatrics; 3:547, 1920.

40. Ely, F. A. & Weingart, J.: J. Iowa S. Med. Soc.; 5:391, 1915.

41. Engering: Zbfl, Bakt. Abt. I, Orig. Bd. 90, 1923.

42. Erb, I. H.: Cited by Rivers, 1922.

43. Finkelstein, H.: Lehrbuch, der Saulingskrankh. Ed. 2, Berlin, 1921, p. 571.

44. Fraenkel, E.: Ztschr. f. Hyg.; 27:315, 1898.

45. Gerbasi, M.: Meningitis from Pfeiffer's bacillus in infant; Pediatria; 32:600, 1924.

46. Ghon, A.: Wien, klin. Wchnschr.; 15:667, 1902.

47. Giese, H. Ugesk. f. Laeger; 75:621, 1913.

48: Grasty, T. S. D.: Am. J. Obst. and Gynec.; 67:1031, 1913.

49.: Greenthal, R. M. & Kelly, G. F.: Influenzal meningitis; Wisconsin M. J.; 23:188, 1924.

50. Hadfield, G.: Case of influenzal meningitis; recovery after repeated lumbar punctures; J. Neuro. & Psycho.-Path.; 3:342, 1923.

51. Haedke: Munchen. med. Wchnschr.; 1897, xliv, 806.

52. Hecht: Jahrb. f. Kinderheilk.; 1903, lvii, 333.

53. Henry: J. Path. & Bact.; 17:174, 1912.

54. Hill, R. B. & Packard, R. J.: Lancet-Clinic; 113:723, 1915.

55. Hills, R.: New York. M. J.; 107:345, 1918.

56. Hogerstedt: St. Petersburg Med. Woch.; 20:123, 1895.

57. Hotz: Jahrb. f. Kinderheilk.; 1919, Bd. 90.

58. Hubschmann: Inder. Med. Gesllsch. zu Leipzig; Ref. M/m W., 1914, No. 31

59. Hymanson, A.: New York M. J.; 90:1268, 1909.

60. Imperatori, C. J.: Laryngoscope; 25:580, 1915.

61. Johnson, C. K.: Arch. Pediat.; 36:82, 1919.

62. Jordan, E. O.: J. A. M. A., 74:623, 1920.

63. Jordan, H. P. B.: Influenzal meningitis; Rhode Island M. J.; 7:8, 1924.

64. Jordan: Textbook Bacteriology.

65. Jordan, H. P. B. & Smith, D. C.: Rhode Island M. J.; 4:125, 1921.

66. Jordan, E. D. & Sharp, W. B.: J. Infect. Dis.; 31:198, 1922.

67. Jundell, I.: Jahrb. f. Kinderh.; 59:777, 1904.

68. Jundell, I.: Hygiea Medicinsk och Farmaceutisk Manadsskrift.; 74:326, 1912.

69. Kitasato: Cited Jordan's Textbook Bacteriology.

70. Kincaid, H. E.: Influential meningitis with added pneumococcal infection; M. J. Australia; 2:146, 1926.

71. Klinger, R.: Vor.-Bl. f. schweiz. Aerzte; 42:1289, 1912.

72. Klinke: Deutsche Med. Woch.; No. 2. 1924.

73. Kotz: B. kl. W.; No. 17. 1921.

74. Kristensen, M. & Christensen, S.: Six cases of influenza meningitis; Acta Med. Scandinav.; 60:1-6, 1924. (French).

75. Lacy, G. R.: Jour. Lab. & Clin. Med.; 4:2, 55, 1918.

76. Langer, J.: Jahrb. f. Kinderh.; 53:91, 1901

77. Leuchtenberger, R.: Influenza meningitis; Munchen med. Wchnschr.; 71:1061, 1924.

78. Levin: Internat. Clin.; Phila., 3:11. 64, 1920.

79. Lisbonne & Leenhardt: Primary acute meningitis with influenza bacillus; Paris med.; 13:47, 1923. Abstract: J. A. M. A. 80:964, 1923.

80. Litchfield, W. F.: Recovery in a case of influenzal meningitis; M. J. Australia; 1:104, 1920.

81. Lorey: Zbl. fur. Bakt.; Ab. II, Orig. Bd. 79, 1917.

82. Manson, R. L.: Meningitis due to Pfeiler's bacillus; Brit. M. J.; 2:83. July 15, 1922.

83. Marcus, J. H. & Crane, B.: Influenzal meningitis—investigation with report of case in infant; Arch. Pediat.; 45:445, 1928.

84. McDonald, C. S.: J. Path. & Bact.; 12:442, 1908.

85. Meunier, H.: Compt. Rend. Soc. de biol. 52:5, 1900.

86. Moody, E. E.: J. Missouri State M. A.: 13:328, 1916.

87. Mya: Riv. clin. Pediat., Firenze; 1:465, 1903.

88. Nabarro & Stallman: Lancet; 2:743, 1924.

89. Neal, Josephine: Arch. Pediat.; 38:1, 1921.

90. Nedelmann, E.: Influenzal meningitis in infants; clinical study; Arch. f. Kinderh.; 86:98 108, 1929.

91. Nyberg, C.: Finska Lak.-Sallsk Handl.; 57:1369, 1915.

92. Oeller: M. Kl. Wchnschr.; No. 44, 1918.

93. Packard, F. R.: Ann. Otol., Rhin. and Laryng.; 25:706, 1916.

94. Pfuhl, A.: Ztschr, f. Hyg., 26:112, 1897.

95. Prasek, E. & Zatelli, T.: Wien, klin. Wchnschr.; 24:932, 1911.

96. Rhea, L. J.: Arch. Int. Med.; 8:133, 1911.

97. Ritchie, J.: J. Path. & Bact.; 14:615, 1910.

98. Rivers, T. M.: Influenzal meningitis: Am. J. Dis. Child.; 24:102, 1922.

99. Ross, A. & Moore, A. E.: Brit. M. J.; 2:1056, 1913.

100. Saathoff: Munchen. med. Wchnschr.; 54:2220, 1907.

101. Sayce, O. A.: M. J. Australia; 16:25, 1911.

102. Schmidt, P. & Weinberg, M.: Influenza bacilli and meningitis; Klin. Wchnschr.; 3:66, 1924. Abstr: J. A. M. A.; 82:665, 1924.

103. Schnyder, W. & Urech, E.: Meningitis from influenza bacillus; Schweiz med. Wchnschr.; 55:549, 1925; Abstr.: J. A. M. A.; 85:552, 1925.

103-a. Signorelli, J.: Proc. La. State Pediatric Soc.. Meeting, April 1929.

104. Simon, G.: Monatschr, f. Kinderh.; 9:549, 1910.

105. Simon, G. L.: Rev. mens. d. mal de l'enf.; 23:271, 1905.

106. Simon and Aine: Semaine med.; 30:513, 1910.

107. Slawyk: Ztschr. f. Hyg. 32:443, 1899.

108. Spillman, F. & Benech, J.: Province med.; 24.433, 1913.

109. Sprigg, W. N.: Am. J. Obst.; 56:467, 1907.

110. Stangl: J. A. M. A., 73:1048, 1919.

111. Stone, C. T.: Texas State J. Med.; 15:318, 1920.

112. Tebbutt, A. H.: M. J. Australia; 1:415, 1914.

113. Templeton, E. R., King, D. S. & McKean, R: Clinical, bacteriologic and pathologic findings in a case of influenzal meningitis; Am. J. M. Sc.; 163:675, 1922.

114. Testivini: Dauphine med., 22:49, 1897.

115. Thomesco and Grascoski: Bull. de la Soc. des Sc. Med. de Bucharest, 1904, No. 2. Abstracted: Rev. neurol.; 13:44, 1905.

116. Tobler, W.: Cor.-Bl. f. Schweiz. Aertze; 47:881, 1917.

117. Torrey, R. G.: Am. J. M. Sc.; 152:403, 1916.

118. Trailescu, I.: Spitalul Rev. Med. 21:474, 1901.

119. Turner, M. L.: Influenzal meningitis; J. Iowa Med. Soc.; 16:490, 1926.

120. Valentine, E. & Cooper, G. M.: J. Immunol.; 4:359, 1919.

121. Williams, E. H.: New Zealand Med. J.: 15:48, 1916.

122. von Gavel, R.: Influenzal meningitis and sepsis with abscess of knee-joint in child aged one and one-half years; Arch. f. Kinderh.; 79:161-170, 1926.

123. Wollstein, M.; J. Experiment. Med.; 14:73, 1911.

124. Wollstein, M.: Am. J. Dis. Child.; 1:42, 1911.

125. Wollstein, M.: Tr. 15th Internat. Congr. Hyg. & Demog.; 2:57, 1913.

126. Wollstein, M.: J. Exper. Med.; 22:445, 1915.

127. Worster-Drought, C. & Quarterly, S. W.: Influenzal meningitis due to Pfeiffer's masillus; Brit. M. J.; 1:416, 1923.

Records of Drs. Bloom and de la Houssaye.

DISCUSSION.

Dr. J. Birney Guthrie (New Orleans): We had an epidemic of meningitis in 1898 when I was an interne at Charity Hospital. We had fourteen cases that died, all of which were diagnosed as influenzal meningitis. Autopsies were held on most of these cases. In the spring, I was transferred to another service, likewise a colored service, and I saw a child three months old with a meningitis which I called influenzal meningitis. The case did not seem to differ very much from cerebrospinal meningitis. This case was seen in 1899. The child got well and I was very much impressed with how it got well. We gave it hot baths. When the child's temperature was continually high, the child received frequent hot baths; and the child got well. In the army in 1917 I used the hot bath with excellent results as a sedative in cerebrospinal meningitis.

Dr. F. M. Johns (New Orleans): My experience comprises about twelve cases in 18 years. Out of that series only one recovered.

Dr. Bloom is to be congratulated on obtaining fresh live serum in which bacteria will not grow.

In company with one or two other gentlemen, whom I see hear tonight, I was astonished recently to have been shown by Dr. C. L. Downman, of Atlanta, Ga., two cases of recovered staphylococcus meningitis. Those two cases were to my knowledge the only two cases that have ever recovered. The treatment was laminectomy of third dorsal vertebrae giving continuous drainage, and the injection of an iodine solution into the carotid artery. This should add another method of treatment to the ones enumerated by Dr. Bloom.

Dr. Johns (after Dr. Lemann's discussion): Since Dr. Lemann has also brought up the Downman cases, I would like to say I have imported

some of the Pregyl's iodine from Germany and would be glad to lend some to anyone.

Dr. John Signorelli (New Orleans): I recall the epidemic of 1928, wherein I had the opportunity to see nine cases. These nine cases, all were treated exactly alike. Eight died, and one little girl of seven years of age made a complete recovery both physically and mentally. No specific treatment was given this particular child—no treatment that was not given the other eight cases that died. Dr. Guthrie will recall he was asked to lend a helping voice in the treatment of these cases.

The particular case that recovered had only one spinal puncture, which served to make the diagnosis. The organisms isolated were B. influenza and staphylococcus aureus, and there was no doubt as to the diagnosis. No further spinal punctures were made because we thought they would not help; we failed to see how the removal of organisms through the puncture could possibly offer relief, or that the temporary pressure reduction would be of benefit.

After convalescence was complete and on her return to school this girl did excellently in her work. Some of the doctors who were present at the meeting of the Louisiana State Pediatrics Society in 1929 may recall the case, which I presented at that time.

I merely bring this to your attention because I am obliged to conclude that because this child recovered completely, we should not be led to believe that we are in possession of a specific treatment for this type of cases. Rather, I am convinced, the occasional case of this type which recovers represents a patient of exceptional individual resistance which itself accomplishes a spontaneous recovery.

On the other hand, the information Dr. Bloom gives us is very valuable because it opens to us another avenue of thought which may in the future develop into something of real value.

Dr. Bloom (closing): I appreciate the discussion concerning the treatment of influenzal meningitis. I am sorry that I have not placed the case of Dr. Signorelli in my series of recoveries, but I do not believe it has been published. However, I will be glad to include it.

I hope no one of you expected that the plan of treatment suggested is a panacea in all cases of influenzal meningitis. As a matter of interest, out of the 23 cases reported only 6 of that 23 recovering received influenza serum.

The method referred to by Dr. Signorelli has given considerable consideration due to the fact that those who have used it believe that by using it they not only relieved pressure but that it served as a drain.

I hope perhaps additional light may be thrown on the subject in the future and as discussed by Dr. Lanford regarding the second case (which happened to be mine also) that child just about showed evidence of recovery when some unforseen accident happened, and for no reason at all the child suddenly died. But so far as meningitis was concerned the child was practically free of meningitis.

HEMORRHAGIC DISEASE OF THE NEWLYBORN AS A CAUSE OF SUDDEN DEATH.

(REPORT OF THREE CASES OF VISCERAL HEMORRHAGES.)*

JOHN SIGNORELLI, M. D.,†

NEW ORLEANS.

Hemorrhages occur in the newlyborn in unassisted as well as in assisted cases of labor, and may be grouped into three general classes, namely: 1, Those depending upon trauma either in part or altogether; 2, Those depending upon constitutional disturbances, as lues, sepsis, leukemia and prematurity; and 3, Those depending upon causes other than, and independent of trauma or constitutional disturbances. These latter cases are generally spoken of as being based upon a hemorrhagic diathesis, and represent the so-called hemorrhagic diseases of the newlyborn. However, as pointed out in a previous communication, "it is obvious that all cases can not be clearly classified, for very often the etiology in a given case represents more than a single factor; thus, in a case wherein exists an hereditary tendency to hemorrhage, trauma so slight as to otherwise cause no appreciable bleeding would here act as the exciting cause of a hemorrhage of considerable extent."

Widespread study of this group has led to the classification of their symptomatology so as to permit an understandable

*Read before the Louisiana State Medical Society, Shreveport, April 29-May 1, 1930.

†From the Newlyborn Service, Hotel Dieu, New Orleans.

grouping of their clinical picture, based upon the location of the bleeding; to a certain degree, to the extent of the extravasation; to a probable prognosis; and finally also, to the adopting of a rational mode of management. From time to time there has been added to the medical literature the experiences of observers in this field so that today most of these cases of newlyborn spontaneous bleeding are correctly diagnosed, and many are successfully treated. Study of the literature on this subject, however, at once leads to the supposition that most all, or all hemorrhages of the newlyborn take place intra-cranially, within the meninges, supporting structures of the brain, or within the brain itself. Much has been written on the subject of intracranial hemorrhage over a period of years, and more particularly since 1912.

That hemorrhages in this type of newlyborns occur in other parts of the infant body and especially within the abdominal viscera has not been very much stressed in recent literature, and one is apt to be misled into the belief that such cases are rare. That lesions of this type do occur with unsuspected frequency, however, has been shown by the investigations of Hedrén in 1000 and of Spencer in 130 autopsies, as quoted by Ehrenfest. They, and others, have found many cases of subcapsular hemorrhage with rupture of the liver, rupture of the spleen, and hemorrhages into the pancreas, adrenals, uterus and testicles of newlyborns.

It is logical to suspect that if a newlyborn possessed of a hemorrhagic tendency is subjected to severe manipulations of any of its viscera or tissues, then the most probable site for bleeding to take place is at the point where most injury to the tissues was inflicted. Thus, in a case of head presentation with prolonged or difficult labor, where molding is severe and overlapping of the sutures occurs; or, where forceps are applied; or, where pituitrin was used in injudiciously large amounts, the most logical site for bleeding is within the skull cavity, where the abnormal increase of pressure injures the soft parts of tissues within and thereby lowers their resistance to the point where the increased coagulation time of the infant blood met with during the third, fourth, fifth and sixth days of life at once starts a small bleeding which persists, if untreated, because of the coexisting increase in the bleeding time of the blood during this period. As the clot increases in size and causes increased injury to the surrounding structures more hemorrhage takes place; thus, a vicious circle is established which, if unchecked by nature or by medical intervention leads to permanent damage if not, indeed, to the death of the infant.

On the other hand, however, cases of breech presentation exert the most direct or indirect injury to the viscera located in the upper end of the abdomen; namely, the liver, spleen, pancreas, adrena's and kidneys. These injuries result from the passive congestion resulting as the fetal blood is backed up into the still unborn portion of the child after the presenting part has made its exit through the pelvic canal and out of the tight vulvar opening, creating a mechanical hyperemia, which in association with any predisposing factor, as prematurity, lues, or hemorrhagic diathesis, leads to hemorrhage into the most vulnerable viscera, which obviously are the larger ones.

The writer wishes to emphasize that, more often than thought, injury to these organs results from trauma incident to undue pressure against the infant's abdomen during frantic manipulations in artificial delivery, and in some instances during efforts at resuscitation. Here it is important to remember that asphyxia, *per se*, causes a lowering of the coagualability of the infant blood, as pointed out in a previous communication.

Though no definitely stated figures are available, observers who have devoted much time to this subject appear to agree that the most commonly affected abdominal vis-

cera, in their order of frequency are: liver, 11 cases collected by Hugo Ehrenfest; suprarenals, 8 cases collected by Hedrén, 1 by Weltmann, 1 by Loeber, and 1 case of bilateral involvement reported by Runyan; pancreases, 1 case reported by Ipsen; and bladder, 1 case reported by Boehi.

There can be no doubt, however, that a number of cases, trivial in their extent, are never diagnosed and go on to spontaneous recovery; nor can there be any doubt that some cases of extensive and fatal bleeding have never been recognized, cases in which the formidable and rapid clinical catastrophe quickly leading to the death of the infant have misled the physician to ascribe death to causes other than hemorrhage. One might state with no injustice to any one, that the diagnosis of hemorrhage was not even considered as a possibility. One of the cases here reported presented a problem in diagnosis which made a correct diagnosis impossible. This infant was apparently doing very nicely up to the morning of its third day of life, when while nursing at the mother's breast as was its usual habit thus far, it suddenly collapsed, became cyanosed rapidly (pallid cyanosis), and in spite of every effort to restore it, expired within 1½ hours. Since up to this catastrophe the infant gave no signs of illness, and further, since no time was permitted for extensive examination, any statement as to a diagnostic opinion would at best have been a pure guess. The post-mortem findings of a huge subcapsular hematoma of the liver with rupture of the capsule at once explained all the clinical phenomena. Evidently there had been a continuous oozing of blood from the liver which accumulated subcapsularly at the base of the organ, producing finally enough pressure to rupture the capsule. It was this particular phenomenon which precipitated the collapse of the child.

The two other cases here reported were diagnosed ante-mortem only because of the autopsy diagnosis of the first case. More detailed accounts of these three cases follow:

CASE REPORTS.

Case 1. Baby G. was born of a multiparous mother on Feb. 19, 1929, in R. O. A. position. Labor was not unduly prolonged nor difficult. It was considered a normal delivery being assisted only by the use of pituitrin. The baby weighed 9 lbs., 12¾ ozs., and after the usual manual stimulations respiration was satisfactorily established, the infant crying lustily. Put to the breast at the usual routine periods the baby nursed very satisfactorily and seemed to be doing very nicely in every respect. On the morning of Feb. 22, 1929, while in the midst of its 6 A. M. nursing with no apparent cause the baby collapsed and fell from the mother's breast to the bed. At the same time it became markedly cyanosed (pallid type of cyanosis), and though relief measures were at once attempted, death took place within 1½ hours.

Because of the rapid development of the clinical course no clinical diagnosis was ventured beyond the consideration of such conditions as enlarged thymus gland, thrombosis or hemorrhage of some type as being among the possibilities.

AUTOPSY.

On opening the abdominal cavity a large amount of blood was found in the peritoneal cavity, which, on close examination was found to be almost filled with liquid blood and clots. A large clot wsa found in the upper right section of the cavity one edge of which was attached to a loose flap of the liver capsule. The capsule was ruptured at the under surface of the organ, and the liver proper was compressed markedly at its under surface.

Both lungs showed hemorrhages into their structure, especially at the innermost angle of the pleural cavity, and involved fully half of the entire lungs.

The heart and all other organs showed nothing abnormal except a moderate amount of pallor.

Case 2. Baby J. S. was born of a primaparous mother on May 29, 1929, in L. O. A. position. Labor was difficult, due probably to the mature age of the mother, and was also prolonged. Pituitrin was used, and later low forceps applied. Baby weighed 5 lbs., 7½ ozs., and respiration was established by artificial aid. It got on quite satisfactorily taking a dextrose formula from the bottle pending the establishment of maternal milk supply. On June 2, 1929, it refused its formula for the first time and examination revealed a pallid cyanosis of moderate degree, and intermittent in type. The respirations were shallow and irregular.

Seen in consultation the same afternoon the baby was found quite pallid, with respiration and pulse indicating the probable existence of hemorrhage. In spite of the usual treatment, including whole blood injections and the application of radium over the spleen, no relief was obtained and the patient died about midnight.

A clinical diagnosis of hemorrhagic disease of the newlyborn with extensive bleeding from a large viscus, most probably the liver, was made because of the rapid and uncontrollable development of a picture of exsanguination, and particularly because of the lesson learned from the autopsy of case 1.

AUTOPSY.

Thoracic cavity: The thymus was moderately enlarged and showed nothing else of note. Several hemorrhages of small size were found throughout the lung tissue in both left and right lungs. The heart was negative. The mediastinum was free from abnormalities. Abdominal cavity: On opening the abdominal cavity there were found hemorrhages into the mesentery; into the kidneys; left adrenal, and large clots as well as liquid blood free in the peritoneum cavity.

The capsule of the liver was ruptured at the under-surface of the organ, and from the dangling portion of the broken capsule portions of blood clots were hanging.

Case 3. Baby J. C., Jr., was born of a multiparous mother on July 29, 1929, in R. O. A. position. Labor was prolonged and associated with wrapping of the cord about the baby's neck. However, the delivery was considered normal, being aided only by the use of pituitrin. Baby weighed 9 lbs., 14 ozs., and was moderately cyanosed. The respiration was soon established by the aid of artificial measures, cyanosis soon disappeared and the baby nursed satisfactorily and did well in every respect until the second day of life, when it was found collapsed and exhibiting a marked pallor.

Seen in consultation on July 31, 1929, this case recalled at once the striking similarity it bore to the pictures exhibited by cases 1 and 2. Clinically it ran a course quite parallel to the other two cases and death followed on the night of July 31, only several hours after the occurrence of the physical collapse. For these reasons a clinical diagnosis of hemorrhagic disease of the newlyborn with hemorrhage of the liver was made.

AUTOPSY.

Thoracic cavity: The thymus was normal. The lungs and pleurae as well as the mediastinum showed nothing abnormal. The heart was normal. Abdominal cavity: On opening the abdomen the entire peritoneal cavity was found filled with liquid blood and several clots. The capsule of the liver was broken at the under surface and a large clot was still partially attached to the under surface of the liver. A small hemorrhage was found in the left adrenal. Two hemorrhages were found in the right ileo-cecal region below the peritoneum. These were about the size of a silver dime and a twenty-five cent coin respectively.

The impressions derived from these cases by the writer are in perfect accord with the expressions of Hugo Ehrenfest, who states that "it is striking that infants, eventually dying from a ruptured liver, apparently as a rule, seem perfectly normal of approximately the first three days of life. Symptoms of serious illness do not manifest themselves until a considerable amount of extravasated blood has reached the peritoneal cavity. Then death is prone to occur suddenly and unexpectedly. Only a post-mortem examination can in these cases reveal the true cause of death."

The three cases here presented emphasize what has been generally observed in this type of cases, namely: that in most of them the bleeding begins by the end of the first forty-eight hours of life, and that, although a few cases of severe hemorrhage do occur as late as from the fourth to the sixth day after birth, these cases of later onset are, as a rule, less formidable in their progress and milder in every phase of clinical behavior. Occasionally, too, cases have been seen in which the coagulation time has been within the normal. Thus, it appears quite clear that there must be other factors connected with blood chemistry and blood physiology, not yet understood, which play quite a direct part in the production of these clinical pictures.

Nevertheless, the writer is of the opinion that, with the contributions of Rodda upon the coagulation and bleeding times in newlyborns as a basis, the routine examination of these factors during the first six days of life in every newlyborn would lead to the recognition of these potential bleeders in the vast majority of cases. Moreover, such efforts might lead to the discovery of other facts and data which would further broaden our knowledge of the subject.

These thoughts, added to the desire to point out the unsuspected frequency of these cases, as proven by the occurrence of three within a single year in his own practice, are the direct reasons for this presentation.

BIBLIOGRAPHY.

1. Ehrenfest, Hugo: Birth injuries of the child. Appleton ,1922.

2. Pfaundler, Meinhard & Schlossman, A.: Diseases of children. Lippincott, 1908. v. 2.

3. Signorelli, John: Intracranial hemorrhages in newlyborn etiology, pathology and symptomatology. New Orleans M. & S. J. 79:448-53, 1926.

4. Loeber, M. & Lawson, E. H.: Hemorrhage of newlyborn. New Orleans M. & S. J. 78:723-26, 1926.

5. O'Donnell, W. S.: Injuries and accidents in newlyborn. Internat. Clinics, 1:48-53, 1924.

6. Abt, I. A. ed.: Pediatrics. Saunders, 1924-25. v. 2.

DISCUSSIONS.

Dr. Maud Loeber (New Orleans): Dr. Signorelli has given us a very timely subject and in his usual excellent manner has covered in a very terse, comprehensive way, not only hemorrhagic diseases of the newborn but also both frank and latent hemorrhages of the newborn. He brings out definitely a very important point, namely, that hemorrhagic diathesis may play a vital part as a contributory if not a causative factor. In making this statement we feel that he must have had in mind the close relationship of visceral hemorrhage, especially of those occurring beneath the capsule of the liver, as well as the inter-cranial hemorrhage of those delivered by cesarean section when he says that "there must be other factors connected with blood chemistry and blood physiology, not yet understood, which play quite a direct part in the production of these clinical pictures," for we do know that babies born of uremic mothers are prone to hemorrhage especially in these locations.

Latent hemorrhage cases seem to be one of the points on which we should agree with him, that there are other factors than that of physiology and of blood chemistry, which we do not particularly understand, that would make us be on the lookout for these latent hemorrhages. Particularly is that true of those hemorrhages occurring under the capsule of the liver, and also intracranial hemorrhages which we see after cesarean section delivery.

In both of those types of hemorrhages, we know that they are particularly associated with cases of uremia in the mother. We have seen blood chemistry done on some of the dead babies after delivery, and the blood chemistry has coincided so closely with the mother's blood chemistry that we feel there are some factors which should be looked into for prognosticating the latent types of hemorrhage before delivery.

I am sorry Dr. Signorelli did not take up more completely the true hemorrhagic diseases of the newlyborn, as I am sure he could give us some very valuable suggestions along those lines, the hemorrhagic disease of the newlyborn where the hemorrhages come from the mucus membrane and are seen some three or four days after birth for apparently no cause. I hope he will say something more on this subject when closing his paper.

With Dr. Signorelli, I agree we should certainly be on the lookout for hemorrhage. Being on the lookout, it would certainly help us in our diagnosis rather than depending entirely on symptoms, because in many of the frank hemorrhages we have active thymus glands certainly simulating intracranial hemorrhages in cyanosis and in convulsions.

I tried to collect a few figures from the two hospitals with which I am connected at home in New Orleans, and I am indebted to Dr. R. R. Roberts and Dr. Jack Strange and Dr. E. Smith for helping me compile the data from the records. From our records in the Charity Hospital of New Orleans, we were unable to get very many of the cases because we filed the baby's record with the mother's. Where the babies die—and that is the point we are stressing in Dr. Signorelli's paper—sometimes the cause of death of the baby is not on the mother's chart and we lose the record. However, since January 30, we were able to find seven cases. I am sure, as Dr. Signorelli has pointed out, there must be a great many more. I am sure I have seen more than that, and I am sure the other visiting men have seen more also.

Of those seven, two were frank hemorrhagic diseases of the newlyborn, and the others were hemorrhages occurring at the time of birth.

We went over the records from Touro Infirmary for the past six years, and in that time there were forty-eight deaths from intra-cranial hemorrhage of the newlyborn. Twenty-one died at birth or a few hours after; six died in twenty-four hours; nine in two days; two in three days; three in four days; one in five days; two in six days; one in eight days; two as late as ten days, and one as late as three weeks. They were all autopsied and diagnosis was made for death.

There were forty-four cases of hemorrhagic disease of the newlyborn. Twenty-three cases died. We are not concerned with those. Of these hemorrhagic diseases of the newlyborn that died, three lived two days; five, three days; three, four days; four, five days; two, six days; two, ten days, and two died in twenty-four hours.

To go back again to the frank hemorrhages, there were ninety-one cases with birth injuries. Fifty-two died. They were all intra-cranial hemorrhages, and most of the intra-cranial hemorrhages occurred after difficult delivery, and the diagnosis of intra-cranial hemorrhage and hemorrhage of the lung resulting from hemorrhagic disease of the newlyborn was made at autopsy.

Dr. Charles J. Bloom (New Orleans): Apropos of the splendid paper that Dr. Signorelli has presented today, it brings back to me an instance occurring not many months ago when I was called in to see a baby who died a few moments after I made my first examination. Not being able to conscientiously diagnose this case, we asked for a post-mortem which was granted, and much to my surprise we found a large amount of extravasated blood in the cortex of the adrenals and in the outer surface of the kidney. This case unquestionably belongs to the group of hemorrhagic diseases of the newlyborn which Dr. Signorelli has called to your attention.

In taking up the question, we have in the past given much attention to the intra-cranial hemorrhages of the newlyborn. The literature has contained many articles since 1912 as to the injuries that the child necessarily encounters in being delivered either due to prematurity, pituitrin, or the like, and we have to some extent disregarded the injuries to which breech presentations may be subjected.

It isn't out of order to remind you that every child is essentially a bleeder, that the new environment brings into play a change of physiological actions and that there must be a readjustment of the red blood cells. We know definitely through the works of Rodda and other men that the total red count and the hemoglobin, the total white count, and the like, are much different a short time after birth than it will be a week or ten days later. For this reason, I imagine in the Old Testament the idea of prolonging the so-called ritual circumcision beyond the seventh day was because they had in mind that the child was essentially a bleeder.

In view of this, I am making it a routine in my private work to have not only a coagulation test made, but in most cases a bleeding time and platelet count, because there are instances where the coagulation may be normal and the bleeding abnormal.

I certainly have enjoyed listening to Dr. Signorelli's paper, and while in a great number of these cases our efforts may be in vain, still, on the other hand, we will be able to classify the case, and where the child has this tendency we may be called upon to do things that may obviate further trouble and, in some cases, death.

Dr. M. Couret (New Orleans): The subject of hemorrhagic disease of the newborn is always of interest to me because of the large number of autopsies that I have the privilege to hold on cases of this kind. It is really appalling the number of deaths in the newborn that is due to this condition. For this reason I have tried to explain to my satisfaction its cause, but with little result up to the present time.

When we use the term "hemorrhagic diathesis," I believe we ought to explain just what we mean. If we mean hemophilia, then we are talking about a deficient condition in the blood vessels, accompanied by a prolonged bleeding time and a prolonged coagulation time, that begins at birth or some future time and lasts through the life of the individual. It is quite true that in the new born both the coagulation time and the bleeding time are prolonged, but between the eighth and tenth day after birth, these return to normal. I do not think, therefore, that it is fair to say that these children should be classed as "hemorrhagic diathesis" because this condition requires a continuation of bleeding, which we are not certain, would not right itself at the proper time.

My experience has been that in true hemophilia, when a puncture is made of a vein or even in the capillaries of the ear or finger tips, bleeding persists sometimes for several hours. I have not observed this in any newborn with hemorrhagic disease nor in the normal child not so affected.

Dr. Signorelli has pointed out that the coagulation and bleeding times are usually very much longer in infants with hemorrhagic disease than in children not so afflicted. I cannot agree with this statement, because I have seen cases of hemorrhagic diseases where the coagulation and bleeding times were no higher than the usual normal rise.

I do not believe that trauma is responsible for this condition because we find hemorrhagic disease in some newborn that have not been traumatized either at birth or during the process of resuscitation, when this becomes necessary. Furthermore, as Dr. Loeber has mentioned, the hemorrhages in these cases are not so commonly found in the head where the injury would likely be greatest, but are found scattered on the epicardium, along the interventricular septum, on the pleura and deep into the lungs, in the adrenals, in the liver, in the spleen, in the mesentery, and in fact anywhere and in any tissue. Certainly, it is improbable that children showing such extensive large and small hemorrhages, could have been so badly handled to the point where so much injury would be produced.

This condition seems to me most likely to result from some malformation or weakness to the vascular endothelium. Whether it is a delayed or

faulty development of these cells only, or a combination of this with an abnormal coagulation and bleeding time and abnormally high blood pressure, requires more thorough study than has been done.

Another question that arises is why should the disease appear after the second to the sixth or eighth day after birth? This may be explained, I believe, from the fact that the right and left ventricles which are practically the same thickness at birth, suggesting that the blood pressure on both sides of the heart are about equal, change after about the second day when the left ventricle shows a greater thickness than the right. This occurs after the closure of the ductus arteriosus and the foramen ovale and would seem to indicate that more pressure is thrown upon the vessels that are fed from the left heart. This pressure is probably more than can be withstood by the "defective" endothelium, and the rupture of the smaller vessels follows.

Dr. T. B. Sellers (New Orleans): This is an interesting and instructive paper not only for the pediatrician, but for the obstetrician as well. Often it is difficult to explain the cause of sudden death of infants five to ten days following a normal delivery. This is especially hard to explain when we consider the number of cases delivered by artificial means, such as version and forceps, without complications. Autopsy is the only means we have of making a correct diagnosis in many of these cases.

The case which Dr. Loeber reported two years ago of hemorrhage from the suprarenal was one that I delivered, a para four, presenting a simple breech delivery in which the baby was apparently normal at delivery.

I would like to stress the value of this routine procedure: the immediate injection of 10 cc. of whole mother's blood in every difficult delivery and a standing order for the administration of whole blood to every baby on the first appearance of unusual symptoms, such as twitching and cyanosis, of course, to be repeated from time to time as indicated.

I would also like to call attention to the fact that the obstetrician can be of great service in assisting the pediatrician in getting an autopsy. At the Baptist Hospital we have endeavored to obtain an autopsy on every stillborn, and, in my private practice, I think I can safely state that we have been able to get permission in at least 90 per cent of the cases.

Dr. J. E. Bailey (New Orleans): As we go along and see these infants, say, forty-eight hours after delivery and we see these symptoms of cyanosis and twitching manifesting themselves, we ought to consider the fact that probably this hemorrhage has been going on for twenty-four, maybe forty-eight, hours before the symptoms manifested themselves. We should be on the lookout for the unusual, or the signs of hemorrhage that we frequently overlook.

We might mention one or two of them. First, would be the good baby, the baby that will remain quietly in his crib without crying. A baby that is described as an unusually good baby during the first twenty-four hours, we are suspicious of. The average baby when stimulated to cry will continue to cry after the stimulus is removed, whereas the baby that is hemorrhagic will immediately stop.

A baby that refuses the breast, that we have a great deal of trouble in getting to take a little water out of a bottle, we can be suspicious of.

Those are probably the two main things to look for when we see that. Get busy right away, and don't wait for three or four days to elapse before going ahead with the treatment that you use.

Dr. John Signorelli (closing): I want to thank the doctors for the liberal discussion. Particularly, I want to thank Dr. Loeber for having gone to the trouble of reviewing the statistics and reemphasizing the fact that these cases are not as infrequent as we have been led to believe.

Dr. Loeber mentioned very correctly the connection that there is in some of these cases with toxemias of pregnancy. The potential cause or the indirect causes that are associated with these cases are not fully understood. And just as the last word has not been said as to the direct cause underlying the hemorrhagic diathesis, so too, the last word has not been said in connection with the many things that might act as indirect causes in producing these clinical pictures.

Dr. Bloom mentioned something about Rodda, and since I referred to Rodda's name I wish to take this opportunity to explain that Rodda's work consisted in investigations of the coagulation time and bleeding time and platelet count in newlyborns during the first six days of life. He found that during this period coagulation and bleeding time, even in normal infants, was increased from the second up to and through the sixth day, after which time there was a return to normal; understand, in the normal babies, babies that did not show any sign or tendency whatsoever to hemorrhage.

He also found in cases which he observed, which later developed hemorrhagic diseases regardless of their location, the bleeding time, the coagulation time were enormously elevated. He reports cases where the coagulation time was advanced as high as forty minutes and the bleeding time ninety

minutes. The platelet counts were also abnormally low in all of these cases.

Dr. Couret mentioned the fact that he has been fortunate in seeing a large number of autopsies. I thought Dr. Couret would say he has been fortunate or unfortunate in seeing the vast amount of bleeding lesions or hemorrhagic lesions in each one of these cases that we have been fortunate enough in getting at autopsy. I, personally, do not feel we should congratulate ourselves on the number of autopsies.

I have not been as fortunate as Dr. Sellers. I can't get ninety per cent autopsies of infants that die in my practice. I have to beg for them and sometimes even make promises which I know are not correct. For instance, in one of these cases reported today, I somewhat bribed the father by telling him maybe I could learn something from this autopsy that might help to save his other baby if he should happen to have another in the future.

Along these lines, I wish to mention that there is some work being done in the United States, both in Chicago and in St. Louis, along the line of the possibility of developing some dietetic preventive measures to be applied to the mother during the period of pregnancy which might prevent the resulting of a hemorrhagic child being born. The work is still in the experimental stage, and although some cases have been reported where mothers thus fed have given birth to children who are not hemorrhagic babies, they are too few to be counted upon with any finality.

Dr. Couret mentioned many little points that would call for so much discussion that I am afraid I cannot go into it. For instance, the real cause of hemorrhagic diathesis. The real cause of the hemorrhagic diathesis is based upon a multiplicity of factors. We are not so much concerned with the academic point of view. We are willing to admit that the surface endothelial lining of the blood vessels may be even more important a factor than the abnormality in the blood platelet count, the coagulation time or the bleeding time.

What we are appealing for here is more attention on the part of the obstetrician, on the part of the pediatrician and on the part of the general practitioner to be on the lookout for these cases. We feel if we do more work and observe more closely all of our newlyborns, we cannot only check up the work of Rodda but perhaps learn new facts, new factors which might lead us to more definite and more tangible information.

MASTOIDITIS AS A CAUSATIVE FACTOR OF ILEOCOLITIS AND BRONCHOPNEUMONIA.*

CASE REPORTS.

J. R. FERNANDEZ, M. D.,

WALLACE, LA.

From time to time since 1921 there have been reported from different sections of the world a sufficient number of series of cases to prove conclusively that mastoiditis and middle-ear infections play an important role in some cases of respiratory and gastro-intestinal disturbances. These infections act either as a focal point, the ear supplying toxins and bacteria through the blood stream and lymphatics, or by direct drainage through the Eustachian tube, and then on to the respiratory and gastro-intestinal organs. Probably in some instances both of these methods are responsible.

The material for this paper is the result of a series of 15 cases treated at the Charity Hospital during April and May, 1929. These patients, upon admission, presented more or less the same pathological triad, namely, gastroenteritis, bronchopneumonia and middle-ear disease, which later proved to be of mastoid origin. These little patients were dangerously ill as evidenced by the rapid respiration, numerous bowel movements and high temperature, which in the majority of cases was septic in character and very high (105°). This combination, resulting in profound toxemia and exhaustion by dehydration from the frequent bowel movements and inability to retain nourishment, proved to be rapidly fatal. The patients were first admitted to the pediatrics service. This department taxed every resource at its command, yet a high mortality ensued until surgical measures addressed toward the middle-ear and its adnexa were instituted.

In order to thoroughly appreciate the exact condition it is necessary that we

*Read before the Louisiana State Medical Society, Shreveport, April 29-May 1, 1930.

refresh our minds and keep constantly before us the peculiar anatomical structure of the middle-ear and its communicating pathway, the Eustachian tube, which in children is short and of a large lumen, infection of which very often is enhanced by the presence of large adenoid growths in the naso-pharynx as well as faucial tonsils. In my mind, the relation of the middle-ear proper to the antrum and the direct communication between the two by virtue of the aditus makes it almost impossible to have a middle-ear infection without some antrum involvement, and especially is this so if the condition is permitted to go on to a spontaneous rupture which is the usual result of earaches from otitis media. This latter anatomical feature is very important to bear in mind at all times, and especially so in those cases where one or more tympanotomies have been done and the ear ceases to drain after a few days. It is always possible in this type of case, when the other symptoms continue unabated, that the mastoid may still be a focal point also supplying organisms through the Eustachian tube. Then, too, it is well to emphasize that infants, that is up to one year, present no mastoid cell development, but every chi'd has at birth a fully developed antrum, which we all know is a large cell in the mastoid bone communicating directly with the middle-ear, and in diseased middle-ear conditions frequently present areas of destruction in the mastoid bone far removed from the antrum. Further quoting from Philipps: "* * * While there are few or no mastoid cells in very young children, the diseased space usually covers a considerable area both in depth and width."

Since mastoid disease is the important feature of this paper, I think it very proper that I mention the objective signs of mastoditis because they are the only means of our knowing when the patient is in need of operative drainage. The drum appearance is not to be depended upon except when there are positive signs of disease, discoloration, sagging perforation, drainage and the usual older signs of middle-ear disease; tenderness and swelling of the mastoid are valuable when present, but their absence means nothing. Many mastoids present no pain or tenderness on pressure, and on all external appearances are normal. Whenever a mastoid presents with the oldtime picture of swelling, pain on pressure and profusely discharging ear, it is no longer a simple mastoiditis, and the patient is a candidate for general septicemia, menigitis, thromboplebitis, or brain abscess. I have seen so many sad results in patients when the surgeon delayed operating, waiting for the usual signs of mastoditis, that I do not think a mention of mastoiditis at any stage is complete without a warning on the points of diagnosis. All that I have mentioned relative to carefulness in diagnosis may seem irrelevant here, while in reality it is one of the big things because our ability to help a mastoid patient of any type in need of surgical interference depends upon a correct diagnosis at an early age.

Now to return to the cases, the subject of this paper. As has already been stated, these little fellows were the victims of a triad highly toxic in character and rapidly destructive to life because of the ravages upon the two vital systems—namely, gastro-intestinal and respiratory, each one responsible for more deaths in children than any single disease. Although these cases proved to be of mastoid origin, there were two distinct types—namely, firstly, those presenting otorrhea at the time of examination, and secondly, those without otorrhea; and yet otitis media, and later mastoiditis, actually existed as proven by exploratory tympanotomy and roentgen-ray examinations.

To further impress you with the seriousness of this type of disease, I wish to mention here that the dead book records show that before any operative measures were instituted no less than 15 patients presenting the same triad were admitted into the hospital and died in spite of all that was done for them. There were some who died within 24 to 72 hours after

admission, while some lingered as long as three weeks; one case admitted in February died in May.

Briefly, the gastric-manifestations varied from a mild to a very severe diarrhea—vomiting, frequent bowel movements—green, watery, and at times bloody. All had some respiratory involvement from a bronchitis to bronchopneumonia in its various degrees of severity. One case by roentgenogram showed an unresolved pneumonia and a collapsed lung.

The temperature was septic in character, at times ascending to 104° and 105,° in the most part remaining 99° to 103° and 104°.

It was evident when everything was considered that the mastoids were supplying toxins to the bloodstream and discharging pus into the Eustachian tube, or both, thereby perpetuating a vicious circle. It is well to mention here that in almost every instance tympanotomies were done, and in some cases daily; in fact, sufficient often to prove to us that drainage through the external ear canal was not sufficient. We were dealing with a deep-seated condition that required extensive free external drainage.

Mastoidectomies were done on all cases from then on, one or both sides as was indicated—only one death resulted; all other patients recovered—some promptly, others less rapidly, while several lingered a long time. The unresolved pneumonia, and even one negro child additionally handicapped with a tubercular cervical adenitis, recovered. The remarkable feature of these cases is that they were operated during the height of the disease regardless of severity of the gastro-intestinal or pulmonary condition, since it was evident that there was little hope for their recovery. They were operated under nitrous oxide and ethylene gas anesthesia. Since that time, and at the present, we are operating this type of case locally.

It follows from the foregoing that any child, in fact anyone, with a condition similar to those I have described who does not respond to the proper treatment or improve as he should, needs repeated and thorough ear examination and requires 100 per cent cooperation of the general practitioner, pediatrician, otolaryngologist, and last, but by all means not least, the roentgenologist, who in many instances will be the only means of clearing up the diagnosis.

CONCLUSIONS.

1. The middle-ear and mastoid have a definite place in acute infections of children in many cases where there is involvement of the gastro-intestinal and the respiratory organs.

2. They also play a double role: (a) Toxic, (b) direct infection by causative drainage through Eustachian tube—or both. In chronic or recurring intestinal toxemias the mastoid may act by focal infection; undermining the health of the child and flares up periodically.

3. Although some authorities advise against operating in the acute stage, we disagree, and it is our conviction that any case showing no improvement after proper treatment for a sufficiently long time, and in which death seems to be the only outcome if permitted to continue, should be given its one chance. If not all, some can be saved even at that last stage. We are reporting only one death in a series of 15 cases.

4. We believe that in doubtful cases in which there is no improvement an exploratory mastoid operation is justifiable. Certainly no harm can be done by scratching the cortex sufficiently deep to investigate.

5. We wish to repeat that the closest cooperation on the part of the general practitioner, pediatrician, otolaryngologist and roentogenologist is an absolute necessity if we hope to help this type of patient; and we should like to warn the pediatricians against placing too much dependence upon their examination of ear-drums because of the reasons set forth in this paper.

I wish to thank Dr. S. B. McNair, Senior Resident E. N. T. Surgeon at Charity

Hospital, who was the first to call our attention to these patients, and my Chief, Dr. Homer Dupuy, under whose active supervision this work was done, also Dr. Amadee Granger, Charity Hospital Radiologist, whose original work on the mastoid, especially in young subjects, is noteworthy, and whose roentgen-ray work was invaluable to us from a diagnostic standpoint.

This paper was offered with two objects in view: Firstly, to place before you the remarkable results obtained in this type of case; and, secondly, to provoke discussion and stimulate interest and work along this particular line. I firmly believe that there is much undone and trust that the remarkable results obtained will encourage others to do some work along this line, and I am sure we will have some interesting reports at some later meeting.

DISCUSSION.

Dr. Maud Loeber (New Orleans): Dr. Fernandez has described so accurately in his paper the typical case of ileocolitis due to the parenteral cause, mastoiditis, that there is nothing one can add to this picture, except to say that he certainly has called our attention to a very important group of cases. We agree with him that our greatest mortality at present is in gastro-intestinal and broncho-pneumonia cases. Dr. W. McKim Marriott and Dr. H. W. Lyman of St. Louis as early as 1924 called attention to these aural focal infections showing that a close relationship exists in infants between a middle ear infection and disturbances of the gastro-intestinal tract. These cases reported correspond in each detail to those seen and treated by Dr. Fernandez, and I wish to emphasize the life-saving measure which the otologists perform in eradicating this foci of infection. From this presentation of his work on ileocolitis due to sinus infection, with the probability of infection in the mastoid, will evolve a great life-saving procedure among infants under one year of age.

Dr. Fernandez is perhaps too modest in taking credit for saving the lives of these infants. The picture he presents of the dehydrated, toxic, extremely ill little patient with severe ileocolitis, associated not infrequently with vomiting and the broncho-pneumonia which as we know is always a bad complication of ileocolitis, respond almost immediately to tympanotomy or mastoidectomy. As Dr. Fernandez very truly says, as soon as the diagnosis is made of middle ear or mastoid involvement, irrespective of temperature and condition of the individual, it is imperative that some operative procedure be instituted. We believe that the high fever and toxic symptoms of the gastro-intestinal tract are reflexly induced by the infection of the middle ear. As he so carefully points out, a draining ear is not at all sufficient proof that the mastoid is not involved, nor is the fact that an ear drum is not bulging the criterion for not performing a tympanum paracentesis for we have seen both conditions improve to uneventful recovery after proper or repeated operative interference was instituted. High persistent temperature with or without explanation for temperature in the lungs, or a stubborn case of gastro-intestinal disturbance, or temperature with inanition unexplained solely by inanition demands the services of the otologists. Not infrequently, as he says, ears have been seen by otologists who have disclaimed any infection in the ear because of the appearance of the membrane and have advised waiting for incision because there was no bulging or other signs present which they seem to feel is imperative before they institute operative procedure.

Therefore when he states that only by roentgen-ray are we sometimes able to incriminate the mastoid in the pictures of gastro-intestinal and broncho-pneumonia cases and this secondary condition clearly brought out, is a point well worth reckoning with. The fact that we see cases of serious infection with broncho-pneumonia, or again cases of serious ileocolitis, does not always lead us to believe that we have a mastoid because these conditions are very often co-existent as a serious broncho-pneumonia or a serious ileocolitis or both may exist without any ear involvement. We frequently have some complications of ileocolitis and not infrequently broncho-pneumonia in cases in which the mastoid was primarily at fault, and vice versa. It is certainly encouraging to the pediatrist to find that a new field is open to lower our mortality rate. We appreciate that mastoids and middle ear infection are important causative factors in raising at present the high mortality figures in broncho-pneumonia and ileocolitis.

A routine examination of infants' ears is as essential in a physical examination as the taking of a temperature or the examination of the chest.

Dr. Fernandez has certainly given us a very practical paper, one which will aid materially in saving the lives of many cases which swell the number of deaths due to ileocolitis and broncho-pneumonia. I wish to thank Dr. Fernandez for bringing out this point so clearly, and outlining the picture so completely, and calling the attention not only of his department, but also the at-

tention of the pediatrists to the part that middle ear and mastoid infections play in infant morbidity and mortality in ileocolitis and broncho-pneumonia.

Dr. M. P. Boebinger (New Orleans): I wish to preface my remarks and congratulate the doctor. The subject is very important. He brings out the anatomy well. If you will take the trouble to read up on the anatomy of the middle ear by most of our otologists, you will find if the question were put to them "What is the anatomy of the middle ear," authorities would tell you it comprises the Eustachian tube, middle ear, and mastoid. Getting back to the subject on intestinal and bronchopneumonia cases, you must keep in mind, therefore, your mastoid and focal infection.

You will find it is very hard at times to make your diagnosis as to whether you have a middle ear infection or not and unless you are very careful, extremely careful, you will pass up this diagnosis for many reasons. First, it is rather hard to handle the very young and the infant. The canal is very small. The osseous formation has not taken place. You are practically dealing with the membraneous canal. Our roentgenologists will assist us very much. Pain on pressure is reliable but if you are in doubt, you can never open a drum too soon but rather too late. Of c urse, the adenoid tissue must be removed as soon as possible, the tonsils as soon as possible.

You must not forget or overlook history or symptoms, a very important thing, especially in your thorax or colitis cases. When dealing with the middle ear, my personal experience leads me to believe a large majority of our middle ear infections and mastoids are secondary. Whether that be due to adenoid vegetation or whether it be due to some influence, as a cold, or whatnot, the mastoid is always a focus that one must be on the lookout for. The size of the mastoid in the infant, which the doctor has brought out so well, is most important as it is small, in fact only an antrum. We know it is undeveloped. No cells —no mastoid process in the infant. It is a muscular development. The antrum you see in the infant mastoid is very large, about the size of an ordinary red bean. It is not so hard to make the diagnosis. Pressing will not help you but when you are in doubt, you are justified in making an exploratory operation. After getting through the skin you will find a very thin cortex.

I have in mind a case in which the history is like this: we had a little ear discharge and the infection had passed from the middle ear to the mastoid and the drum and middle ear cleared up, looking perfectly normal. I did a mastoidectomy. The case cleared up.

Dr. Monte F. Meyer (New Orleans): Dr. Fernandez has had wonderful results, fourteen recoveries with one death but I want to sound a warning. I don't think just because you have a case of middle ear abscess you ought to go into the mastoids. Ileocolitis and bronch-pneumonia and the middle ear diseases are certainly a triad, but still there are a lot of cases that will get well if you watch them and open up their ears. In a series of over a hundred cases of middle ear disease (not the triad type) last year in private practice, we didn't have a single mastoid. I do not believe you see in private practice the same type (ileocolitis and broncho-pneumonia) that you see in Charity Hospital. We certainly must admit that we are seeing more and more types of severe infection of middle ear disease.

It is just like going to the hospitals in New York and Boston some years ago, and then every case of running ear turned out a mastoid operation but they have gotten away from it, and Dr. Lyman of St. Louis in the last convention at St. Louis said that he is not even operating on as many of these children (of the triad type) as he did when he first started this work. He believes he gets better results if he waits until the acute state is over, give them a chance to build up.

Dr. Granger has recently found a new line in the roentgen-ray examination of infants mastoids under 3 years of age. It ought to be of help to all men doing mastoid work. It has been helpful to me in the last two cases (one case I will cite). I had treated a little child, three years of age that had repeated attacks of earache with frequent tympanotomies but still I wasn't satisfied that the child was doing as well as it should. I thought it had a double mastoiditis. It was just this particular line of Dr. Granger that was negative, that made me defer operative interference and the ears cleared up.

Dr. C. A. Weiss (Baton Rouge): I want to thank Dr. Fernandez for bringing up this important subject. It goes to show that the influence of the infection of the special sense organs is much greater than we give it credit for being. However, there are several features about this paper that are sort of doubtful in my mind. I will have to deliberate on them before I can accept them.

And there is this question: "Which was here first, the hen or the egg?" Doctor mentions the triad; he mentions the gastro-intestinal conditions first, respiratory system second, and the mastoid condition third. Does he put the mastoids third and last because it fits there, or it is a causative factor in this particular condition? If the infec-

tion comes from a mastoid does it enter the digestive and intestinal track direct from the nasophroynx or is the intestinal condition an end result of the toxemia produced in the blood by the infection? We have quite a number of these mastoid developments in the la grippe and influenza infections, without any intestinal pathology.

The fact of operating on the patient at the height of infection is an important point brought out and while it would be permissable, in probability much more easily accomplished in hospitals than in private practice, I think. We would experience considerable difficulty if we advise operation on a patient in the extreme stage of ileocolitis and at the height of the disease. However, it is a valuable point that the doctor brought out that there wasn't a fatality in this series of cases even though operated on at the height of disease.

Dr. W. P. Coyle (Orange, Texas): This has been so interesting to me that I am going to take a little of your time. The doctor's paper was so well prepared that it could not help but interest anyone who is present at a meeting of this kind. It was especially interesting to me because I agreed with so much of what the doctor told us, and more interesting because there were some points with which I did not agree. It is such a daily condition that we have to face and one of so much importance and on which so much can be said that in entering a discussion of this paper, it is difficult to know just what points one may touch upon and still keep within the limit of time. I assume that there are many general practitioners here, and that they recognize the fact that we have thousands of cases in place of a few cases of indigestion in infants, which are caused by middle ear infections.

My experience does not agree with the doctor's that there are many of these cases that are difficult to diagnose. I think many, regarded as chronic cases, are recurrent cases simply because these children in this civilized age are taught to blow their noses. We have many large Eustachian tubes in children who have a recurrent otitis media every time they have a coryza if they are permitted to blow their noses and their ears are not strongly reinforced by finger pressure at external meate. There never was a child who would blow his nose until someone taugh him to do it.

The chief difficulty with the general practitioner, unless he is modern, is that he overlooks the middle ear infection, because in fully 50 per cent I believe 75 per cent, there is no pain, no appearance of discomfort of the ears to direct his attention to these infections, another is that he seems mindless of the fact that there are no moving parts of the middle ear, and fills them full of oil and other things that are difficult for the specialist to get out when he sees them.

Another point that the general practitioner is frequently seriously misled by is the fact that these ears have ruptured spontaneously or maybe draining as a result of a previous myringotomy, but there is no relief of symptoms because they are draining under just as much pressure as many which are not draining at all. When the tympanum is dried and inspected, no opening will be disclosed, there will be no visible aperture. But with gentle suction or observation for a few minutes, a pinpoint opening through that drum will be revealed through which secretions force their way. I think that radical procedure in these cases, at least that is my experience as a country specialist, is greatly overdone. The cases that baffle us and lead us into serious difficulty are the rapidly fulminating acute cases that are due to virulent infection, and come to us like a storm out of a clear sky. We will also see these cases at times in adult patients.

The other type that frequently comes to us in a hopeless condition are the chronic otitis media cases that have had a sudden obstruction. It is my observation that fully 90 per cent of mastoids which are treated as emergency cases, in which there is plenty of temperature, pain and redness, with either a drum that has not drained or a drum that has drained under pressure and which when they are freely opened, that though apparently in extremis, will be on the royal road to recovery within 24 hours.

Dr. Fernandez, Wallace, La. (closing): I think the best part of my paper was the discussion. These cases were presented with two objects in view: first, to acquaint you with this type of case and put before you our remarkable and marvelous results; and second, to promote extensive discussion. I do not think this series of cases needs any further defense; the patients are alive and can testify to their results, while the records of the dead book of Charity Hospital speak for the poor unfortunates who were not operated.

As to Dr. Meyer's statement, that we do not see these cases in private practice, I disagree. We should see them, and the fact that these cases are not made in the Charity Hospital proves that this type of disease does exist in private practice at some stage or the other. A statement to that effect, namely that they are only seen in the Charity Hospital, is an open confession of neglect. We all know that the Charity Hospital of the city of New Orleans is the clearing house for the State of Louisiana, and it is here that the numerous mistakes are all washed clean. It rarely

happens that a patient comes to Charity Hospital without first having consulted a physician, and in numerous instances more than one, and at times one or more specialists. Dr. Meyer further states that we operate too often, and he mentions the numerous cases of mastoiditis he has seen in private practice during the last year or so. My paper does not deal with all cases of mastoiditis in infants, but only those as are associated with ileocolitis and broncho-pneumonia. Dr. Meyer has not stuck to the subject of this paper. In further answer to his charge that we operate far too often, I will state that we only operate in this type of case, as was clearly brought out in my paper, after all other measures have failed and death seems to be the only outcome. I would like to remind Dr. Meyer of the remote dangers in this type of case where there has been no surgical interference, namely: hopeless deafness in later life, and as we see every day, patients presenting in early youth with brain abscess, thrombophlebitis, meningitis and other disastrous conditions.

Now as to Dr. Coyle's statement, I must disagree with him in part. He seems to take the same attitude as Dr. Meyer with reference to operating too often or too early. Dr. Coyle can not testify as to the safety of these little patients until he has followed them fifteen or more years of their life. He may find in some of these apparent cures, some brain abscesses or some hopelessly deaf. I have in mind at this time a patient recently in our service who was sixteen years of age; his history was a middle ear infection in early infancy which had cleared up for a time, but later manifested itself with a persistent discharge. This patient paraded from clinic to clinic in the great city of New Orleans for fourteen years in a vain effort to cure his discharging ear. Finally he was admitted to the Charity Hospital—an acute case with temperature 105°, septic, dizzy and complaining of terrific pains in his head. And do you know that with the history of fourteen year discharging ear and the condition which he presented at the time of admission that they made a roentgenogram of his sinuses and overlooked the mastoid completely? It proved to be a case of thrombophlebitis and brain abscess, and we have succeeded, after surgical interference, in undoing within a few days what stumbling blindness on the part of the doctors took fourteen years to develop. Gentlemen, conditions of this kind are nothing less than a shameful disgrace.

Now I think we have just scratched the surface of this subject, and I hope that there are those among us who will go home and operate these little fellows who do not do well early, and I promise that you will save many a life and probably avoid the development of serious trouble in later life. With reference to operative consent on the part of a parent, I have never had any trouble along this line, and I suggest in this case that the doctor explain fully the present condition and present in much detail the remote possibilities as I have just described.

Dr. Coyle's idea of recurrent otitis media being caused by children blowing their noses is very amusing and suggests a probable cause for this recurrent condition. However, I do not think that occurs very often because of the fact that an infant is not prone and does not know how to blow his nose until he has reached an age of about four years. Again, I want to impress you with the fact that we do not operate all cases of otitis media and mastoid just because they are cases of otitis media and mastoid. Every one of the cases in this series was operated only after definite signs of mastoid destruction were found by roentgen-ray and examination, and only as a last and heroic measure when every other procedure had failed. I have in my mind right now another patient who had been treated at Touro Infirmary, a baby sixteen months old, treated for recurrent enteritis with otitis media, an infant sixteen months old weighing thirteen pounds. He was sent to Charity Hospital, they could do nothing for him. This infant presented temperature 105°, vomiting, unable to retain nourishment. After establishing the diagnosis of mastoiditis a bilateral operation was done, and two days later, for the first time in three weeks, the baby was able to retain nourishment. This is just another one of the same type of case that we have presented in my paper.

One feature and characteristic of mastoids in infants that was not brought out, and which was purposely held out until the last for the reason that I want you to carry it home with you, is this: There are those who state and argue that we should not operate because there are no mastoid cells in infants. The antrum is an air space but is not suspended in the air; it is surrounded by bone, and is a part of the temporal bone. The mastoid process later develops from the petrous and squamous portions of the temporal bone. Now, if you will examine the skull of an infant you will find that the sutures and fissures are not completely ossified, but remain loosely articulated and offer a most inviting opportunity for the advance of infection once planted in the mastoid antrum. This latter anatomical characteristic of an infant's skull, together with the soft, spongy structure of the bone itself in my mind is responsible for the prevalence of mastoiditis in infants as compared with the disease in adults.

I wish to thank Dr. Loeber for her kind discussion of my paper. Dr. Loeber's personal contact and association with these little patients as a pediatrician and her keen observation were responsible for our great success along this line, for it was Dr. Loeber who recognized the necessity of doing something else after all other measures had failed.

PARANASAL SINUS DISEASE IN INFANTS AND CHILDREN.*

J. D. SIMMONS, M. D.,

GUNNISON, MISS.

This disease is often a cause of obscure fever and is frequently overlooked by the physician, yet it is so common that a discussion of the subject is considered timely.

The order of sinus development as pointed out by J. Parsons Shaeffer is as follows: "The ethmoid sinuses and antra are present at birth; the latter lie internally to the orbit. They increase in size through pneumatization of the maxillae and are fully developed, extending below the nasal floor, about the age of thirteen years. The sphenoids appear immediately after birth and reach adult size about the age of seven, when the frontals begin to appear as offshoots of the ethmoid cells into the frontal bones. They are fully developed during the thirteenth or fourteenth year, although an indentation of the sinus into the fronal bones may be recognized at the age of three or thereabouts. The ethmoid cells, anterior and posterior, and the antra are most frequently involved in infancy and childhood, due to their early embryological development. Later on in the same order of sequence the frontals and lastly the sphenoids are affected."

ETIOLOGY IN GENERAL.

Paranasal sinus disease occurs as a primmary condition and may also be secondary to, or a coexistent factor with, numerous diseases, such as influenza, scarlet fever, whooping cough, measles, diphtheria, tonsiltis, and pneumonia.

The most common source of infection is from diseased tonsils and adenoids. Not only do they infect the sinuses by direct contiguity of tissue and through the lymphatics, but they also mechanically cause infection of the sinuses, acting as a trap for secretion which is driven backward into the nose from the nasopharynx. Adenoids in this way may keep a mass of secretion constantly in the upper part of the nose, which being stagnant, would soon become mucopurulent and infect the sinuses.

Other etiological factors are exposure, damp, changeable climate with high annual rainfall, inherited tendencies, deficient and improperly balanced diet, poor hygienic surroundings, including improper clothing and ventilation, lack of sunshine, allergy malnutrition or any metabolic disturbance which tends to lower the child's resistance.

Sinus disease occurs most often in the fall, winter and spring months, but it is also seen in the summer months. In the summer it is often caused by swimming. Swimming causes a shrinkage of the nasal mucous membranes and virulent bacteria from water or from the bather's own diseased tonsils, adenoids or other tissues, reach previously inaccessible parts of the accessory sinuses. This, combined with the chilling of the body and lowered resistance, favors infection.

SYMPTOMATOLOGY IN GENERAL WITH DIAGNOSIS.

Paranasal sinus disease is divided into acute, sub-acute and chronic sinusitis. As mentioned above the acute infectious diseases are usually complicated with an acute sinusitis. Other forms of acute sinusitis may vary from an acute coryza or rhinitis, frequent and prolonged, especially in the winter months, to an acute condition which resembles influenza. The fever may be high in the acute type, respiration is increased, general malaise and cough are evident. Most of the time there will be an enlargement of the anterior or

* Read before the Section on Medicine at the Sixty-third Annual Session of the Mississippi State Medical Association, Vicksburg, May 14, 1930.

posterior group of cervical glands. Headache is a constant symptom in older children and is relieved with onset of free nasal discharge, either through anterior nares or naso-pharynx or both. The temperature usually falls with the appearance of the discharge, which may be purulent, mucopurulent or bloody. Epistaxis may occur, and there is usually much sneezing and sniffing. The sneezing is most marked in the morning.

The subacute or chronic type of sinusitis gives an entirely different picture. The child on first appearance is below normal, underweight, poorly nourished, usually a mouth breather, anemic, nervous, and irritable. He is forgetful; and if he is of school age, the teacher will complain that he is inattentive and does not concentrate. Often the mother will say, "Doctor, my child picks his nose all the time, grits his teeth and groans in his sleep. I believe he is wormy." Don't let the mother's diagnosis influence your treatment. Go into the past history thoroughly and see if the child does not have repeated head colds, with nasal discharge, vertigo, headache, breathing through the mouth, a non-productive cough, early morning sneezing, or frequent spells of earache.

On examination, some of these "wormy" children will have some of the above mentioned symptoms, and in addition you will find crusting around the anterior nares, or a mucopurulent discharge with excoriation of the lips, inability to breathe through the nose because of enlarged and diseased adenoids. On throat examination you will find diseased tonsils, hypertrophied patches of lymphoid tissue on the posterior wall of the nasopharynx, thickening and congestion of the posterior pillars, with some evidences of mucoid or mucopurulent discharge down the nasopharynx. There is usually an enlargement of the anterior or posterior group of cervical glands. The child will run a low-grade fever, rarely above 99.4° unless there is an acute exacerbation.

Some of these children have had their tonsils and adenoids removed without complete relief of symptoms. Sometimes the remnant of adenoid tissue hypertrophies and a second operation is necessary.

Unilateral discharge from the nose should suggest to the physician foreign bodies. If some of the above symptoms are found after ruling out foreign bodies, syphilis and tuberculosis, one may feel satisfied that he is dealing with sinus disease.

Direct examination of the nose after shrinkage will often reveal pus or mucopus coming from under the middle turbinate.

Transillumination and careful roentgen-ray plates are other methods that help in the diagnosis.

COMPLICATIONS.

The complications of the sinus disease, both acute and chronic, are numerous and directly or indirectly affect different parts of the body. Some of the most frequent complications are: Laryngitis, pyelitis (acute and chronic), bronchitis, bronchiectasis, otitis media, facial erysipelas, marginal blepharitis, conjunctivitis, keratitis, corneal ulcer, orbital cellulitis, with edema, of lids, arthritis (acute and chronic), nephrosis, acidosis, diarrhea with dehydration, endocarditis, myocarditis, deafness, and chorea.

TREATMENT.

The vast majority of the cases fall into the hands of the general practitioner or pediatrician, but treatment rightfully belongs to the rhinologist.

A great many cases get well without any treatment; many of them develop chronic sinusitis and receive no medical care other than the usual home remedies.

The proper supervision of sinus disease calls for the closest cooperation of mother, physician or pediatrician, and rhinologist, and divides itself into dietetic, climatic, medical, and surgical treatment.

Dean and Daniels proved that a diet rich in carbohydrates and lacking in Vitamine

A is conducive to the development of sinus disease. The child should have an abundance of cod liver oil, butter, and succulent vegetables, as a proper diet is essential. Sunshine, ultra-violet rays, proper ventilation of living and sleeping quarters and clothing to suit the changeable weather are beneficial measures.

Removal of diseased tonsils and adenoids breaks up a vicious cycle, and some authors claim that as high as 90 per cent of cases are cured by this procedure.

For the acute case, rest in bed is essential. Mild calomel catharsis and shrinkage of the nasal mucous membrane with ephedrine, one per cent, either with nebulizer or by medicine dropper every three hours, followed by mild suction, will hasten the drainage and normal ventilation of the sinuses.

For the chronic case of means, removal to a dry climate will usually clear up the sinuses.

Other surgical measures, such as antral lavage, nasoantral opening and more radical procedures, are sometimes necessary.

CONCLUSIONS.

1. Sinus disease is often the cause of obscure fever, and is a very common condition in infants and young children.

2. Paranasal sinus disease in infants and children can be the focus for a large number of secondary manifestations.

3. Removal of diseased tonsils and adenoids benefits all and cures a majority of the cases of sinus disease.

4. Active cooperation of the rhinologist, pediatrician or physician, and mother is necessary for the treatment and cure of sinus disease in infants and young children.

PARTIAL BIBLIOGRAPHY.

1. Brown, J. McKenzie: Sinusitis in childhood. Ann. Otol. Rhin., and Laryng., 38:500-505, 1929.

2. Dean, L. W.: Paranasal sinus infection in children. J. A. M. A., 93:838, 1929.

3. Dean, L. W.: Paranasal sinus disease in infants and children. J. A. M. A., 85:317, 1925.

4. Hasty, Frederick E.: Paranasal sinus infection and swimming. J. A. M. A., 89:507, 1927.

5. Lynch, R. C.: Sinus disease in infants and children. New Orleans M. & S. J., 82:336-338, 1929.

6. Mitchell, T. C., and Shea, J. J.: Nasal sinus disease in children. Arch. Pediat., 39:230, 1922.

7. Spake, Laverne B.: Paranasal sinusitis in children. Kansas City Clin. Soc. Bul., 4:20-23, 1928.

8. Weinstein, M. A.: Sinusitis in infancy and childhood. M. J. & Record, 130:95-98, 1929.

9. Gutteridge, Eric: Paranasal sinusitis in children. M. J. Australia, 496-500, 1927.

Due credit is given to W. F. Prior Company, Inc., Hagerstown, Md., for selection of some of the reference material.

DISCUSSION.

Dr. J. W. Gray (Clarksdale): This is one of the most valuable and practical papers we have had, and there is little to add to what he has said. One point I would like to stress is the extreme value of proper diet. Most of these children I think belong to the general practitioner during the first year—I do not agree that the nose and throat man should have charge of them at all. I have not observed much benefit from local treatment. I think generally diet and hygiene will do more. I think any operation on the sinuses or throat is a mistake. If they are properly nourished, especially so far as vitamins A and D are concerned, nearly all will get well without surgical interference. They should be given lots of cream. The diet should be increased two or three times. If they cannot get cream, give a good cod liver oil, not the irradiated kind so much used now—that does not contain vitamin A and is of no value in these cases. Ultraviolet irradiation and sunlight will do a great deal more for them than anything else, at least I have found it true in my practice.

Dr. G. L. Arrington, (Meridian): I disagree with the speaker—you do have to treat them. The general plan outlined is very good. Cod liver oil is most important. Most children, however, will vomit it or refuse to take it. For older children, 10 years of age or more, tablets are the best form, and it will absolutely build up their resistance. Transillumination will not always work. Where you have a transillumination which is not positive, and the throat man says it is all right, and you do not know what is the trouble—take a roentgen-ray. It is much more satisfactory than transillumination in the child. These cases we get in little children where the doctors say to take them off proteins and fat and keep them in bed, if you will have a roentgen-ray made, keep them on a high caloric diet with cod liver oil, you will cure them in much less time.

SINUS DISEASE IN GENERAL MEDICINE.*

MONTE F. MEYER, M. D.,

NEW ORLEANS.

Why should sinus infection interest the general practitioner? A large number of diseases have their origin in affections of the accessory nasal sinuses. Dr. Joseph Evans in a recently comprehensive discussion of the subject enumerates 37 well established general diseases with their origin largely from this source. Chief among the diseases, listed by Dr. Evans, which arise from focal infection are myocarditis, endocarditis, arthritis, pyelitis, acute rheumatic fever, affections of the skeletal muscles, eye disorders, asthma, bronchitis, bronchiectasis and gastro-intestinal disorders.

The occurrence of such complications is so much dependent on the anatomy of the sinuses that a brief description is at least necessary for an understanding.

What are the sinuses? What are their functions?

They are cavities in the bones of the skull lined with mucous membrane with openings into the nose. To keep healthy they must have proper ventilation and drainage for their natural secretions.

The function of the sinuses is to make the bones which contain them both lighter and stronger, so that most of these bones will crush before they break. They also separate the nose from the delicate structures of the eye behind, and from the cranial cavity above. Because of the large number of cells which go into their make up, the sinuses provide a means whereby air is gradually moistened and heated by a slow interchange. They are also of prime importance in voice production, and may be likened to the sounding box of a musical instrument, thus giving resonance, the essential element of a good carrying voice.

The sinuses are divided into two groups:

(1) Anterior. Made up of the maxillary antrum, frontal and anterior ethmoid.

(2) Posterior. The sphenoid and posterior ethmoid.

The former group have their openings into the nose through the middle meatus below the middle turbinate. The posterior group is located in the superior meatus or above the middle turbinate. Therefore, when either of the groups is affected., it is the location of the pus in the nose which may give a clue to the offending sinus.

What is the cause of sinus infection?

It is the result of some local disturbance, such as deflected septum, hypertrophied middle or inferior turbinate, or anything which prevents proper ventilation and drainage in the face of an acute nasal infection.

There is probably never a case of influenza which does not involve the nasal sinuses. This fact must be remembered for when a patient convalescing from influenza runs a long, continuous, low grade temperature without any other explanation, the sinuses should be carefully examined.

Which sinuses are most frequently involved?

The anterior ethmoid and antrum are in equal proportion, then the sphenoid and frontal to a lesser extent. Antrum diseases in about 30 per cent of the cases are due to dental infections either by contiguous or blood stream infection.

What symptoms or diseases should the medical man think of as being associated with sinus disease?

(1) Headaches. (2) Head pains. (3) Ocular diseases. (4) Focal infection. (5) Respiratory infections. (6) Gastro-intestinal disturbances. (7) Anemia.

*Read before the Louisiana State Medical Society, Shreveport, April 29-May 1, 1930.

(8) Cardiac infections. (9) Neurological. (10) Endocrine disturbances.

Headaches and head pains are so closely associated by patients, that they are unable to differentiate them. When there seems to be no relation of the headache or pains to the use of the eye or to the state of the gastro-intestinal tract or in the case of women to the menstrual period, but the patient gives a history of repeated colds or tells of pains dating back to a cold associated with a post-nasal discharge or running nose, then the sinuses should be thought of as the offender.

A further clue to the offending sinus especially in acute infections, is the location of the pain and the nature and the character of the pus. Frontal sinus headaches usually have a true periodicity, starting around 10:00 A. M., lasting until 12:00 M., then ceasing and beginning the next day at practically the same time. The pain is more marked over the frontal region and trans-illumination may show some interference with light transmission.

In antrum infection the pain is greater over the antrum and cheek bone but may be frontal or supra-orbital. It lacks the periodicity of the frontal and often keeps the patient awake at night. The pain may be referred to the teeth, mastoid or occiput. The pus in acute maxillary sinusitis is usually canary yellow in color and is often expectorated in large quantities two or three times a day instead of in a constant discharge. In latent cases of antral disease, lipoidal injections may have to be resorted to for further differentiation.

In headaches of ethmoidal infection the most prominent symptom is constant pain that is referred to the region between or behind the eye. Use of the eye muscles aggravate it and tenderness is present below the inner canthus. In acute infections the eyelids or entire face may be swollen, these symptoms have to be differentiated from those in acute orbital cellulitis. The pus is watery and constant in acute inflammation.

The sphenoidal type is the hardest type of all to diagnose. The pain is usually in the occiput, but may be in the parietal or temporal region, or even extend to the shoulder, and through the arm down to the finger tips. In the process of an acute cold there may be temperature and a headache so intense as to be suggestive of meningismus or even meningitis.

The chronic inflammations are still further classified according to the pathology as to hyperplasia, empyema or chronic inflammation. It is here that a roentgenogram is essential for a diagnosis. In passing let me state that Dr. Amedeé Granger is the father of sinus roentgenograms in this country. He is able to tell implicity as to the pathology existing in the sinus from the roentgenogram alone.

Nearly every form of ocular disease can be attributed to sinus disease. The most important is retro-bulbar neuritis, in which there is sudden loss of vision, usually monocular in which opthalmoscopy is normal. The sinuses most involved in this type are the sphenoid and posterior ethmoid cells. Other eye diseases that occur are blepharitis, chronic conjunctivitis, phlyctenular conjunctivitis, various forms of corneal diseases, iridocyclitis, vitreous opacities, orbital cellulitis and paralysis of the fourth and fifth nerves.

In many cases of error of refraction, especially in young people, who never can accustom themselves to the glasses, the eyes themselves are not at fault, but rather some sinus disturbance is usually in the background.

Wonderful results have been reported by many American otolaryngologists in the restoration of vision in retro-bulbar neuritis by opening of the sphenoid and posterior ethmoid sinuses.

It has been agreed by Drs. Masher, Skillern, Hurd, Beck, Fenton, and Lynch that it is the duty of the rhinologist to operate on these sinuses in cases of sudden blindness. They all agree that no harm can

ensue if proper surgical judgment and technic is used.

In focal infections the nasal accessory sinuses should be considered as the third factor, teeth and tonsils preceding it. The gastro-intestinal tract, gall-bladder and prostate follow in the order stated.

In respiratory infections the most frequent are chronic bronchitis, bronchiectasis, and bronchial asthma. It is believed that pus from the sinuses is aspirated into the lungs during sleep. Many chronic cases which have lasted for years, some even being diagnosed pulmonary tuberculosis, have cleared up when the associated sinus disease cleared up. Bronchial asthma is in some cases due to a hyperplastic ethmoiditis of a chronic origin. Unless there is a clear cut sinus picture, nasal operation should rather be discouraged than encouraged in asthmatic conditions.

The gastro- intertinal symptoms may be either acute or chronic gastritis, or mucus colitis due to the swallowing of pus. This is especially true in chronic ethmoid infections.

Dr. Silverman in a personal communication tells me that these cases of chronic gastritis are on the increase and are never cured until the sinuses are cleared up.

Anemia of the secondary type is caused by sinus disease, just as any other toxemia.

Cardiac infections such as myocarditis, endocarditis, and arrhythmias may be due to sinus diseases, especially a low grade sphenoditis.

Neurological complications and insanity have been found to be due to sinus disease. In such cases, patients have had almost miraculous cures by the opening of the offending sinus. This relationship seems to have been a little exaggerated in some of the reported cases.

Then too endocrin disturbances may be traced to the sinuses. The proximity of the pituitary gland to the sphenoid sinus makes it involved at times and its dysfunction produces similar dysfunctions in some of the other endocrin glands.

CONCLUSIONS.

(1) The nasal accessory sinuses have a real relationship to many diseases and must be considered by every one practicing medicine whether he is a general practitioner or a specialist.

(2) No examination is complete without an examination of the sinuses, and if the history is at all suspicious, there should be a trans-illumination and roentgen-ray confirmation.

(3) Investigative work on infections of the sinuses has just begun; medical treatment is constantly being improved, and the research work on the infection of sinus and its relation to diet has just begun. Likewise order is taking the place of the surgical pandimonium that has reigned.

(4) Sinus disease is increasing in this climate and even children are becoming more susceptible. The erroneous ideas that the lay person has of sinus diseases and their incurability with operation should be corrected. The whole problem of sinus disease cannot and should not be undertaken by the rhinologist and the otolaryngologist alone but he needs the help of all of his brothers in medicine.

DISCUSSIONS.

Dr. Val Fuchs (New Orleans): Dr. Meyer has brought to your attention a focus of infection that is very often entirely overlooked by the general practitioner.

It is surprising how little knowledge of the anatomy and functions of the sinuses some men have. It quite frequently happens, that when every source of possible infection has been treated without benefit to the patient, that the rhinologist finally sees the case and makes a diagnosis of sinus infection.

If we would remember, that any so-called simple head cold, lasting over 10 days to two weeks, means a sinus infection, we would not see all the sequelae of the chronic infections that we now daily encounter.

While headache is a common symptom of sinus disease, it is not constantly present and we do see far advanced cases of chronic trouble

without any history of headache or any complaints from the patient, attributable to the sinuses. Fairly constant symptoms are frequent head colds and post-nasal dripping and whenever a patient gives a history of such conditions, they should at once put us on guard.

We often hear patients and even doctors say that once anyone starts having these sinuses operated upon they are never finished. This is entirely, an erroneous impression. It is humorously stated, that the otolaryngologist has two methods of treatment: the squirtive and the operative. We believe we go a little further and have a third method, namely, the curative.

Except in the hands of the most radical men, all sinus infections do not mean operations, and when we do resort to surgery, these operations are often of the simple variety in preference to the radical work formerly done. In other words, we do not destroy our normal anatomy to reach the diseased part as often as we formerly did.

It is not always necessary to have pus present in the nose to establish focal infection in the sinuses. The chronic hyperplasias can as readily produce systemic diseases from absorption as the true prevalent conditions. The laboratory has definitely proven that the products of hyperplastic infections, when injected into animals, produce the same reactions as the true pus.

Gastro-intestinal conditions are not infrequent. Some months ago, an elderly woman was referred to me by a general surgeon with a history that for years she had been constantly losing weight, with intermittent attacks of nausea and vomiting. As she expressed it, "she could not keep anything on her stomach." On examination a definite maxillary sinus or antrum infection was found and following operation, her symptoms immediately cleared up and all stomach disturbances ceased. This was purely a case in which the constant swallowing of pus left the stomach in a state of turmoil. This is not an unusual case and we all see this variety quite frequently.

Recently we saw a case, referred to us, for instillation of lipiodal into the lungs, to confirm a diagnosis of bronchiectasis. Examination disclosed a bilateral maxillary sinusitis and without operation and merely by treatment, all symptoms, including a violent persistent cough and chest rates cleared up.

You are all familiar with the cases of asthma that are greatly benefitted by the treatment and eradication of a long standing ethmoidal or maxillary sinusitis. Dr. Dowling, of Rochester, N. Y., in an article, some years ago, stated that he has checked violent acute attacks of asthma, by his method of placing tampons in the nose.

Neuralgias of the fifth nerve, particularly that type which the late Dr. Sluder called "upper half headaches" are involvements of the spheno-palative ganglion from an infection of the posterior ethmoids or sphenoid sinuses.

Again, I say, Dr. Meyer has called your attention to a rather neglected area to think of when looking for focal infection and if you will keep sinus infections in mind, this will undoubtedly rebound to the good of your patients, and many obscure cases will be cleared up.

Dr. Cecil Lorio (New Orleans): I suppose it is really not within my line to appear on the floor now, but I left the medical session for the purpose of hearing this paper on sinus disease. I was disappointed in not hearing Dr. Meyer mention anything about sinus diseases in children.

Dr. Val Fuchs has just made a statement that any chronic running nose can be considered as sinusitis. I suppose there is no type of individual in the practice of medicine that would be more common than the infant or child. It is my purpose principally to ask questions—just to tell me something about sinus diseases in children and infants. I have repeatedly examined them for sinus diseases, unsuccessfully in most cases. I have had assistance, I have had eye, ear, nose and throat specialists to help me, and it is only rarely that we really find the actual pus of sinus disease. I would like to know whether it is possible, or whether with extra technic it is possible, to determine when you have sinusitis, sphenoiditis, or whether it is parasynovitis in children.

Again I would like to ask what would be the treatment for any one of the sinusitis cases in children, because operative procedure in infancy is rather something that the family objects to—those two weeks old I should like to ask.

Dr. Meyer (closing): Dr. Lorio asked how we could tell sinus diseases in children. In addition to the local symptoms of the chronic running nose bilateral over a period of time without any inclination to stop by itself, then roentgenograms are taken and the ethmoids are seen in the roentgenogram from birth on, the antrums around the age of 2 years, and the frontals around 6 years, the sphenoids between 2 and 6 years. Only last week I made a roentgen-ray examination of a child 3 years old and showed antrum disease almost positively in this way. The treatment in young children is to use nasal packs of 10 per cent argyrol, and if tonsils and adenoids are enlarged to remove them. It is surprising how well these children tolerate nasal packs that are left in for an hour with a piece of adhesive tape in front of their nose so they will not slip out, but remember to tell the parents of the child's affected sinuses and that it takes a long time to get well. In antrum operation on a child, the only thing necessary is a naso-antral opening made with a Douglas puncher, and enlarge with small Wagner puncher.

NEW ORLEANS
Medical and Surgical Journal
Established 1844

Published by the Louisiana State Medical Society under the jurisdiction of the following named Journal Committee:

H. B. Gessner, *Ex-Officio*
For one year: S. M. Blackshear, M. D., H. W. Kostmayer, M. D., Chairman
For two years: W. H. Seeman, M. D., Randolph Lyons, M. D., Secretary
For three years: John A. Lanford, M. D.

EDITORIAL STAFF

John H. Musser, M. D.....*Editor-in-Chief*
L. S. Lippincott, M. D.....*Editor*
Willard R. Wirth, M. D.....*Editor*
H. Theodore Simon, M. D.....*Associate Editor*
Frank L. Loria, M. D.....*Associate Editor*
D. W. Jones, M. D.....*Associate Editor*
H. L. Rush, M. D.....*Associate Editor*

COLLABORATORS—COUNCILORS

For Louisiana	*For Mississippi*
H. E. Bernadas, M. D.	J. W. Lucas, M. D.
Daniel N. Silverman, M. D.	L. L. Minor, M. D.
C. C. DeGravelles, M. D.	M. W. Robertson, M. D.
J. B. Benton, M. D.	T. W. Holmes, M. D.
C. P. Gray, M. D.	D. W. Jones, M. D.
John McKowen, M. D.	W. G. Gill, M. D.
D. C. Iles, M. D.	E. M. Gavin, M. D.
G. M. G. Stafford, M. D.	J. W. D. Dicks, M. D.
	D. J. Williams, M. D.

Paul T. Talbot, M. D.....*General Manager*
1430 Tulane Avenue

SUBSCRIPTION TERMS: $3.00 per year in advance, postage paid, for the United States; $3.50 per year for all foreign countries belonging to the Postal Union.

News material for publication should be received not later than the twentieth of the month preceding publication. Orders for reprints must be sent in duplicate when returning galley proof.

THE JOURNAL *does not hold itself responsible for statements made by any contributor.*

Manuscripts should be addressed to the Editor, 1430 Tulane Avenue, New Orleans, La.

LOUISIANA MORTALITY STATISTICS.

During the year 1929, according to the figures just released by the Department of Commerce, there occurred 24,723 deaths in Louisiana, as contrasted with 24,987 in the previous year. There were three diseases which showed a marked increase in 1929 over 1928. The influenza death rate was 1,707, whereas in 1928 it was as low as 1,304. Parenthetically in conjunction with a large number of patients who died from influenza, it might be interesting to speculate on how many patients with influenza actually did die, as a result of this disease, how many of these cases would have been called influenza had there been some positive laboratory method of diagnosing the disease, and how many of them represented fever from some other cause. Diseases of the heart increased from 3,721 to 3,965, and meningococcus meningitis from 24 to 65. It is most encouraging to note that typhoid, pneumonia, measles, whooping cough and other more or less controllable diseases had decreased considerably since 1926. There was an increase in the number of accidental deaths, 1,505 people dying as a result of accidents, 190 of which were due to accidental falls, and of these probably 175 represented unfortunate bath tub skids.

As in previous years, diseases of the heart led all other causes of death. One wonders how frequently heart disease was put down when there was apparently nothing which could explain the exitus of the patient. Nephritis was the next great cause of death, with a total number of 2,227, the next most frequent cause being tuberculosis, 1,807, and then pneumonia, 1,741. Influenza is another disease well up in the thousands, 1,707 deaths being reported, almost ten times as many deaths as any contagious and infectious disease except tuberculosis. Cancer, cerebral hemorrhage, and congenital malformations and diseases of early infancy are well up in the thousand mark. From these common causes of death, there was quite a drop to the next cause in frequency. Diarrhea and enteritis of children caused 667 deaths, with syphilis the next great cause, accounting for 555 deaths in the State of Louisiana. We are glad to say that the number of homicides diminished from 406 to 345, although there was an increase of 25 deaths from automobile accidents, 373 people having been removed from this terrestrial sphere this way.

EDEMA IN NEPHRITIS

Some recent work of Van Slyke and his co-workers which has appeared in several Journals has substantiated a conception that was advanced by Starling thirty-five

years ago concerning the mechanism of production of edema. The distinguished English psychologist showed at this time the osmotic pressure of the plasma protein to be about 35 mm., between arterial and venous blood pressure. He pointed out that the osmotic attraction of the proteins in the plasma counterbalanced the hydraulic pressure in the capillaries that would naturally drive the blood fluids out of the capillary bed into the tissue spaces. Consequently, if the concentration of the protein in the plasma diminished excesses of fluid would go into the body tissues.

During the course of the degenerative types of nephritis, some of which are spoken of as the nephroses, there is a very large amount of albumin excreted through the kidneys. As a result of the loss of this albumin, there occurs correspondingly a diminution in this substance in the plasma. Van Slyke and his associates show that when the total protein, normally averaging seven per cent, obtains a figure of from 5.2 to 5.8 per cent, or the albumin, normal figures of which are 4.3 per cent, falls to 2.3 per cent edema developes. The globulin seems to have very slight effect because of the fact that albumin has an osmotic pressure which is four times greater per gram than that of globulin.

Other factors play some part in the production of edema in nephritis, such as variations in salt intake, the effect of fever, disturbances of the heart and circulation and so on, but undoubtedly the hydropigenous effect of the plasma protein lack explains in a very large measure the edema that develops during the course of the nephroses in which there is not a concomitant heart lesion but merely degenerative disease of the renal tubules.

THE NEW HUTCHINSON MEMORIAL BUILDING.

The excercises on the night of December tenth, when the New Hutchinson Memorial Building was formally opened, marked an epochal event in the Medical School of Tulane University, and one which should be of importance to the medical profession of New Orleans and Louisiana. The new building, made possible by a contribution of $1,250,000.00 from the General Educational Board of the Rockefeller Foundation, will be utilized for the carrying out in every way the purposes of the bequest of the late Mr. Joseph Hutchinson. It will be devoted to the care of the ambulatory sick and the injured and at the same time will be utilized for the purpose of instruction of medical students in a manner which is both unusual and without precedent. Each student will be given his own office, wherein the working arrangements will resemble closely those of private practice. Here he will examine and treat patients assigned to him under the supervision of an instructor. There are 105 such offices in the new building under the different divisions of the medical school. Each of the offices is equipped with small dressing room, examining table, desk, laboratory table with equipment, a centrifuge, and a wash-stand. In addition to this novel pedagogic arrangement there are other laboratories where diagnostic and therapeutic procedures more complicated than routine office procedures may be carried out. There are also laboratories for the staff, teaching rooms, conference rooms, an auditorium and other facilities which will go to make a most complete medical school.

On the first floor of the building, quarters have been assigned to the Louisiana State Medical Society. A large, bright, sunny and cheerful secretaries room is part of the suite which contains as well the office of the Secretary-Treasurer and the Editor of the New Orleans Medical and Surgical Journal. The Secretary of the Orleans Parish Medical Society also has an office in this same suite, and adjoining it is a large board room furnished magnificently by the Orleans Parish Medical Society. The Library of the Orleans Parish Medical Society is just above the offices of the State and Parish Societies which are connected with it by a private stairway and small private elevator. The library of the Or-

leans Parish Medical Society and the Tulane Medical School Library, while housed in the same stack room are filed separately. They are under the care of the same library force so that duplications are avoided in both the book files and current medical journals. In this way there is splendid opportunity for enlarging the Library which already is the largest in the South and which has possibilities of being made one of the largest in the United States.

Members of the Louisiana State Medical Society will be welcome at any time in the new domicile of the organization, and the Library facilities of the Orleans Parish Medical Society and Tulane Medical School may be used whenever members of the organization will have need of its facilities.

The close relationship of this new Hutchinson Memorial building to the Charity Hospital, the propinquity of the Hotel Dieu and the Eye, Ear, Nose and Throat Hospital make for a potential hospital center in which many of the members of the Society already carry on the bulk of their medico-charitable activities as well as some of their practice in those hospitals accepting private patients.

HOSPITAL STAFF TRANSACTIONS

CLINICO-PATHOLOGICAL CONFERENCE, OUR LADY OF THE LAKE SANITARIUM, BATON ROUGE, LA.

The first of a series of Clinico-Pathological Conferences was held at Our Lady of the Lake Sanitarium of December 2, 1930. The following physicians were present: Drs. T. S. Jones, L. J. Williams, C. A. Weiss, Jas. M. Adams, W. K. Irwin, U. S. Hargrove, W. H. Cook, W. Pipes, T. C. Paulsen, T. J. McHugh, H. T. Nicolle.

The following cases were presented and discussed. Both the gross and microscopical pathology were shown.

Case 1.—Recurrent sinuses of the chin due to abscess of teeth; by Dr. T. S. Jones; Case 2.—Sarcoma of the leg, by Dr. W. K. Irwin; Case 3.—Papillary cyst-adenoma of the ovary; Case 4.—Colloidal carcinoma of the breast; Case 5.—Membranous enteritis following appendectomy; Case 6.—Subperiosteal abscess of lower end of femur following trauma.

The meeting was presided over by Dr. T. S. Jones, the Chief of Staff and Dr. L. J. Williams acted as Secretary.

Case 3.—Papillary cystadenoma of the ovary.—Mrs. K., aged 21, admitted October 29, 1930, discharged November 9, 1930. Irregular menstruation. Chief complaint was biliousness, headache and pain in right lower quadrant, from which the preoperative diagnosis of chronic appendicitis and right cystic ovary was made following a physical examination. Patient was operated on October 29, 1930. Ovary and appendix removed. Recovery uneventful.

Laboratory Examinations:—Urine, trace of albumen; Blood; WBC 9200, L 31, M6, P 59, E 3, B 1.

Tissue:—Appendix showed chronic inflammatory changes. Ovary measured 5x4x3 cm. There was a cauliflower-like excressence through an opening of the sac where the ovary was separated from the broad ligament. On section the ovary appeared to be completely filled with a gray white papillomatous growth with the spaces filled with a mucoid material. The capsule was very thin measuring about 1mm. thick. The microscopial examination showed the wall to be composed of connective tissue with very little ovarian tissue. The central mass showed a typical papillomatous mass which was covered over with one or more layers of ciliated epithelium which was thrown into folds.

Case 4.—Mrs. E. E. C., age unknown. Patient old and talks incoherently. No personal history could be obtained. Patient admitted September 24, 1930, discharged October 7, 1930. Working diagnosis: cystic tumor of breast.

Physical examination was negative except as follows:—Tumor the size of an orange in outer area of left breast. Near the nipple there was fluctuation while deeper there was felt a hard mass which was not adherent to the underlying tissues. No axillary glandular enlargement.

Laboratory Reports:—Urine, faint trace of albumen. WBC 6500, L. 38; M 5; P 54; E 2: B 1.

Preoperative and post operative diagnosis was cystic degeneration of the breast.

Tissue report:—Breast measures 12x10x7 cm. Near the nipple the skin was discolored and there was fluctuation. No fluid could be aspirated through a 20 ga. needle. On sectioning the breast there was found a large cavity near the nipple. This was filled with a colloidal fluid. Deeper and under the fluid there was a mass measuring about 2.5cm. in diameter. This was hard and mottled white and gelatinous. In the colloidal material were white masses which were composed exclusively of carcinomatous cells. The tumor showed typical carcinoma areas interspersed with areas of colloidal degeneration.

Case 5.—Mrs. A. G., white, aged 53 years. Admitted November 13, 1930, died November 25, 1930. Patient was operated on October, 1929, for hernia and was advised to return for a laparotomy and plastic. On admission patient complained of pain in right lower quadrant of abdomen. Appetite poor. Bowels constipated. Physical examination disclosed tenderness in right lower quadrant; small, retro-placed uterus causing a sense of bearing down upon standing. Otherwise negative. On November 17, the patient was operated upon. The appendix was removed and the uterus suspended. On November 18 the patient's temperature rose to 103½°. On November 20 there was copious diarrhea, nausea, frequent urination. Temperature 98°. Pulse hardly perceptible. From this time until November 25 the temperature was from 98° to 96°. Pulse 84 to 68, weak and barely perceptible.

Laboratory reports:—Urine faint trace of albumen, otherwise negative. Stools contained a large amount of blood after November 19, with a very foul odor. Blood: 11/13 WBC 5500; L 32; mono 5; P 58; E 1; B 1; myelocytes 3. November 19; WBC 3000; L 38; Mono 8; P 54.

An autopsy was held on November 25, the same day of death. On opening the abdomen there was found about 500 cc of slightly cloudy fluid. Several of the loops of the small intestines were found matted together with a recent exudate. The area involved began about three inches from the ileo-cecal valve and extended in the ilium about eighteen inches. This part of the intestine was very hyperemic as was also a portion of the omentum. On opening of the intestine there was a dirty gray membrane which was rather firmly adherent and on removal left a raw surface. Microscopic examination showed that there was a necrosis of the entire mucosa and in some places of the submucosa. The external coat was apparently normal. The liver showed a chronic hepatitis. The kidneys showed an acute nephritis. Spleen, chronic splenitis. The site of operation showed no infection nor was there leakage from the appendiceal stump.

VICKSBURG SANITARIUM AND CRAWFORD STREET HOSPITAL.

Staff Meeting, December 11, 1930.

Abstract: Intestinal Obstruction Complicated by Pregnancy.—Dr. J. A. K. Birchett, Jr.

Patient: Colored, female, aged 32 years; housewife; three children; admitted to hospital November 19, 1930.

Chief Complaint.—Unable to have bowel movement for one week. Began with pain in abdomen and desire to defecate. Was given several doses of castor oil and salts and an enema, all without results. The same day began to vomit and has vomited off and on since that time. For past three days vomitus has had a foul odor. Three days following onset family doctor was called and made a diagnosis of locked bowel and advised operation, going so far as to make arrangements for the patient to be sent to the hospital. However, the pain became less and vomiting ceased for 48 hours, the doctor having given her a hypodermic, presumably morphine. Two days later the pain again became severe and gradually increased in intensity until patient readily consented to come to hospital for operation.

Past History.—Had operation last year about this time for low abdominal pain and was told that she had a pus tube and appendicitis. Tube and appendix were removed. Has had three normal deliveries, the children living and doing well. No history of digestive, circulatory, respiratory, or renal disease. No tuberculosis or cancer in family. Menstrual history not abnormal except for excess of pain in the right lower quadrant; usually regular; three to seven days' duration. Has not menstruated for the past five months.

Family History.—Mother, aged 58 years, and father, aged 60 years, living and well. Three sisters and two brothers living and well.

Physical Examination. Temperature 100°F.; pulse 140, weak; blood pressure 100/80; respiration 22. Slender, of medium height, complaining of severe pain in abdomen and appearing to be acutely ill. Tongue coated; breath foul; heart rapid, regular. Abdomen distended; tender throughout. Midline scar of previous operation, well healed; no hernia. Symmetrical tumor of

lower abdomen extending as high as the umbilicus, consistent with a fibroid or pregnancy. Peristaltic movement is noted in the distended loops of the intestine and the pressing against the abdominal wall. At the upper end of the old scar, the patient complains of intermittent but acute attacks of pain. Pelvic examination reveals soft pregnant cervix; no evidence of beginning of labor.

Blood: Leukocytes, 5,000; differential leukocyte count, small lymphocytes 11, large lymphocytes 6, polymorpho-neutrophiles 83, with 58 immature forms; hemoglobin 87; red cells 5,000,000; no malaria found; Wassermann, Kahn, and Kline and Young tests negative. Urine—negative except for a few pus cells.

Procedure: A diagnosis of acute obstruction with pregnancy was made and an immediate laparotomy advised with a poor result in view as the patient had now been obstructed for one week. After passing the duodenal tube and washing the stomach thoroughly of fecal contents and the administration of 1,000 cc. of glucose solution intravenously, the patient was sent to surgery. Under spinal anaesthesia, using 150 mg. of novocaine, an exploratory incision was made parallel with the old incision. As the peritoneum was opened, there was a gush of free peritoneal fluid but no evidence of any ruptured intestine. At the upper end of the old scar, a loop of ileum was completely shut off by the pressure of the large pregnant uterus against the adherent loop and there was some recently formed adhesions between the bowel loop and the uterus. After separation of these loops and severing of the bands the continuity of the bowel was established. On further search, another band was found occluding the terminal ileum, this band stretching from the region of the appendix to the surface of the mesentery. When this was freed, all collapsed bowel became filled and though there was some interference with the blood supply of several inches of the ileum, hot packs soon restored the circulation and active peristalsis was noted over the distended loops. With this evidence a contemplated enterostomy was not done and the abdomen was closed with the patient in a serious condition though not markedly more so than before operation was begun.

Progress.—First 48 hours were very stormy but continuous lavage of the stomach, removing the regurgitated intestinal contents and 2000 cc. of glucose solution daily and with spontaneous abortion on the night following operation, there was rapid improvement after the third day. On the third day the patient had a large free bowel movement. Patient discharged as well on the fourteenth posto-perative day.

TRANSACTIONS OF THE CHARITY HOSPITAL SURGICAL STAFF.

The regular monthly meeting of the Charity Hospital surgical staff was held on Wednesday, November 19 last. After the usual order of business several interesting cases were presented. A case of fistula in ano, operated upon by Dr. F. L. Loria, and developing an extensive ischio-rectal abscess on the opposite side some six days after the operation, and at a time when the patient appeared well on her way to recovery showed several interesting points. The abscess dissected its way out toward the outer surface, then under the labia majora of the same (right) side, and an extensive cellulitis and lymphangitis extended up onto the right side of the abdomen. The fever was septic in type and continued so after the institution of drainage. After several negative cultures the last was finally positive, showing the streptococcus hemolyticus in the blood stream. The patient died of a profound septicemia some six days after the incision and drainage.

Dr. Ambrose Storck, of the Surgical House Staff, next presented a case of aneurism of the aortic arch on whom he did a "decompression" operation, according to the technic recently advised, creating an arterio-venous aneurism between the right carotid artery and vein. Dr. Storck pointed out that this case showed considerable improvement since the operation, being now free from pain, whereas before the operation it was frequently necessary to give him opiates. Over a short period of time however the thrill appeared to disappear entirely. There was definite evidence of the vessels being thrombosed at the time the case was shown by Dr. Storck.

At the next regular meeting held on December 17 last, five cases of variable interest were presented and discussed. The first was a case of Ludwig's angina which died on the operating table before an abscessed tooth could be extracted and

following the administration of ethylene anesthesia. As the surgeons operating upon this case were not present nothing of particular value was brought out in the discussion. A case of strangulated hernia operated upon by Dr. Mattingly and dying on the night of the eighth post operative day, evidentally of a pulmonary embolism, was presented by the operator. In the brief discussion Dr. Loria brought out the fact that most cases of pulmonary embolism, perhaps 75 per cent of them, died within a very short period of time after the onset of the attack—as a rule within five to ten minutes. The subject of embolectomy in these cases, as recently recommended by Gunnar Nystrum, of Upsalla Sweden, was gone into by Dr. Loria, who also added that such a procedure is at this time impracticable in Charity Hospital. The third case was that of granuloma inguinale in a negro female, who died a short time after cauterization of the area. Dr. R. L. Gordon, in discussing the case, brought out the fact that the coincidental administration of neosalvarsan and tartar emetic was rather a dangerous practice because these individuals have all they can handle taking care of one poisonous metal at a time. This opinion was concurred in by Drs. Reed and I. M. Gage. A fourth case operated upon by Dr. C. J. Miller proved to be interesting. This was a case of rather intractible vesico-vaginal fistula, upon whom an hysterectomy and then a transplantation of both ureters into the sigmoid were performed. The case did nicely for four days following which time she developed a high fever frequently preceded by chills, the patient dying on the eighth day after operation. Dr. Gage, in his discussion, suggested that possibly the transplanting of one ureter at a time might prove more successful, and dwelt some time on the careful preparation of these patients. The fifth case was that of an elderly female 72 years of age who was operated upon by Dr. F. L. Loria for a suspected malignancy of the gall-bladder. The operative findings consisted of extensive adhesions extending from the gall-bladder over onto and ensleeving the second portion of the duodenum, producing a stenosis of the duodenum in this region. The adhesions were broken up and the gall-bladder removed. The patient did nicely for nine days, at no time being nauseated, nor did she vomit, when through a misunderstanding the sutures were removed and an attempt was made to pass a duodenal tube, causing the patient to gag strenuously and to eviscerate. Although repair was performed within 20 minutes she went badly on the fourth day after the repair and died. There was no discussion of this case.

Frank L. Loria, M. D.

MEETING OF THE FRENCH HOSPITAL STAFF.

A regular meeting of the French Hospital Staff was called to order Friday, November 28, 1930, Dr. M. J. Lyons presiding. Those present were: Drs. H. B. Alsobrook, J. N. Ane, C. J. Brown, E. N. Haller, F. Gallo, P. Graffagnino, M. Lescale, L. Hanckes, M. J. Lyons, S. C. Lyons, M. L. Stadiem and E. L. Zander.

The reports of discharges and deaths for the month of October were given by the secretary. The only death of interest was a case of septicemia and staphylococcal meningitis. This case was presented by Dr. S. C. Lyons and opened to general discussion.

The committee on Cesarean Sections reported that several hospitals of the city required a consultation to be held with a Gynecologist or Obstetrician of some reputation before a cesarean section could be done these institutions also had rules and regulations concerning this subject. A motion was made to accept the committee's report and to give it more time to draw up such rules and regulations as the members saw fit for the French Hospital. The report of the Membership Committee was also accepted.

Several suggestions from the Inspector for the American College of Surgeons were read by the secretary. It was decided to have a place alloted on the regular program for the presentation of interesting cases or specimens, and also to purchase the Minute Book and Application Forms for Membership as suggested by the Inspector.

Two moving pictures entitled "A High Posterior Gastroentererostomy" and "Colle's Fracture" were shown through the courtesy of the Petrolagar Company.

There being no further business the meeting adjourned.

Edwin L. Zander, M. D.

TRANSACTIONS OF ORLEANS PARISH MEDICAL SOCIETY

TRANSACTIONS OF ORLEANS PARISH MEDICAL SOCIETY.
CALENDAR.

January 5—**Orleans Parish Medical Society.** Annual Installation meeting, 8:30 P. M.

January 5—Eye, Ear, Nose and Throat Hospital Staff, 8 P. M.

January 9—Physiology Seminar, Tulane Medical School, 5 P. M.

January 9—French Hospital Staff, 8 P. M.

January 12—Sectional meeting, American College of Surgeons.

January 13—Baptist Hospital Staff, 8 P. M.

January 14—Touro Infirmary Staff, 8 P. M.

January 15—Eye, Ear, Nose and Throat Club, 8 P. M.

January 15—New Orleans Hospital Council, Eye, Ear, Nose and Throat Hospital, 8 P. M.

January 16—Physiology Seminar, Tulane Medical School, 5 P. M.

January 20—I. C. R. R. Hospital Staff, 12 Noon.

January 20—Charity Hospital Medical Section, 8 P. M.

January 21—Charity Hospital Surgical Section, 8 P. M.

January 26—**Orleans Parish Medical Society,** 8 P. M.

During the month of December, besides the regular meeting of the Board of Directors, the Society held two special meetings.

On December 2, a joint meeting of the New Orleans Gastro-Enterological Society and the Orleans Parish Medical Society was held. Dr. Frank Smithies of Chicago was the guest speaker.

December 12 the fifth Stanford E. Chaille Memorial Oration was held in the New Hutchinson Memorial Building. Dr. Irving S. Cutter, Dean of Northwestern Medical School, was the orator.

Saturday, December 13, the annual election of Officers for 1931 was held in the office of the Society. The following candidates were elected:

President—Dr. Emmett Irwin.

First Vice-President—Dr. E. L. King.

Second Vice-President—Dr. Louis Levy.

Third Vice-President—Dr. Waldemar R. Metz.

Secretary—Dr. H. Theodore Simon.

Treasurer—Dr. John A. Lanford.

Librarian—Dr. Daniel N. Silverman.

Additional Members—Board of Directors—Dr. C. Grenes Cole, Dr. W. P. Gardiner, Dr. J. D. Rives.

Following the election, the annual dinner of the Society was held at the Chess, Checkers and Whist Club. The attendance was good.

The Society has invited the American Society of Tropical Medicine to meet here next year with the Southern Medical Association.

Longer Life Week was held December 8-13. Dr. Frederick L. Fenno was Chairman of this Committee. The members participated by making speeches before Luncheon Clubs, Schools, Universities and Co-operative Clubs.

The President appointed the members of the Women's Auxiliary as the Committee of Arrangements for the Installation Meeting.

DR. NIX HONORED.

It is a very real pleasure for us to announce that one of the Board of Directors of the Orleans Parish Medical Society has been honored most highly at the recent meeting of the American College of Surgeons. Among the five hundred candidates submitting histories and case reports to be acted upon by the Committee on Membership, those sent in by Dr. Nix were held to be the most complete, most thoroughly worked up and best prepared. In recognition of this the College awarded to him a life membership in the organization. This is a splendid recognition of the doctors of the South. Dr. Nix not only has brought credit upon the South, but has also added luster to the New Orleans doctors, as we members of the Orleans Parish Medical Society are able to shine in the reflected light of a splendid accomplishment.

TREASURER'S REPORT.

Credits	$2,157.60
Expenditures	1,191.23
Actual Book Balance	$ 966.37

LIBRARIAN'S REPORT.

During November, 100 books were added to the Library. Of these two were received by binding, 9 by purchase, 78 by gift and 11 from the New Orleans Medical and Surgical Journal. New titles of recent date are listed below.

The Library will be moved into the new building during the first week of December. Great care is being taken to transfer the collection shelf by shelf in exact rotation in order to avoid confusion and be settled in our new quarters as soon as possible. The library will be closed to borrowers and readers during the moving process, the Medical School Library caring for all calls on those days.

NEW BOOKS—NOVEMBER.

Williams—Obstetrics. 1930.
Stedman—Practical Medical Dictionary. 1930.
Cecil—Textbook of Medicine. 1930.
Jellett—Manual of Midwifery. 1929.
Marfan—Rachitisme. 1930.
Rolleston—Internal Medicine. 1930.
Williams—Minor Surgery and Bandaging. 1930.
Young—Handbook of Anatomy. 1930.
Lambert—Histology and Microscopic Anatomy. 1930.
Hartridge—Histology. 1930.
Tracy—Basis of Epilepsy. 1930.
Grasset—Les Centres Nerveux. 1930.
Boyd—Introduction to Malariology. 1930.
Tubercle—Vol. 2-9. 1920-28.
Young—Treatment of Infections with Mercurochrome. 1925.
U. S. Medical Service—Aviation Medicine in the A. E. F. 1925.
U. S. Labor Dept.—Family Status of Bread Winning Women in four cities. 1920.
Pelnar—Das Zittern. 1925.

H. THEODORE SIMON, M. D.,
Secretary.

AMERICAN BOARD OF OBSTETRICS AND GYNECOLOGY.—The American Board of Obstetrics and Gynecology, composed of nine members and examiners, elected by The American Association of Obstetricians, Gynecologists, and Abdominal Surgeons, The American Gynecological Society, and the Section on Obstetrics, Gynecology, and Abdominal Surgery of The American Medical Association, was formally organized in Niagara Falls, September 16, 1930. The function of the Board is to grant certificates indicating proficiency and specialization in Obstetrics or Gynecology, or both, to those who comply with its requirements:

This Board has been in the process of organization since 1927. It puts into action a determined effort on the part of these three national organizations to improve the standards of practice of obstetrics and gynecology. It expects to accomplish this by various activities, such as the investigation and encouragement of graduate extension study facilities and active clinical assistantships for men desiring to specialize in these branches, and it will endeavor by regular examinations to determine the competence of specialists in obstetrics in obstetrics and gynecology who apply for the certificate. Certain outstanding specialists will be granted certificates on the basis of their attainments alone, but only by a vote of the entire Board after recommendation by the Committee on Requirements. A second group is asked to undergo a practical clinical examination, whereas a third and younger group has both written and clinical examination and must submit records of a group of cases in order to qualify.

The Board does not intend in any way to interfere with or limit the professional activities of any duly licensed physician, but it does aim toward standardized qualifications for specialists in obstetrics and gynecology.

The first examination for candidates will be held simultaneously in nineteen different cities of this country and Canada on Saturday, March 14, 1931. Detailed information and application blanks may be secured from Dr. Paul Titus, Secretary, 1015 Highland Building, Pittsburgh, Penna.

NEW PSYCHIATRIC DIRECTORY—In 1928, the second edition of the Directory of Psychiatric Clinics for Children in the United States, was published. This directory included, in addition to a list of clinics offering psychiatric service for children, a brief description of any psychiatric services conducted by state departments, a list of state hospitals and institutions for mental cases, and a statement of the activities of state and local societies for mental hygiene. This material is now being again revised and brought up-to-date. In addition to what was obtained in 1928, material will be gathered relative to psychiatric clinics for adults.

The success of this compilation depends largely on the cooperation and assistance of the directors of psychiatric clinics. The gathering of data relative to psychiatric clinics for adults is especially difficult, as no recent directory of such clinics is available.

LOUISIANA STATE MEDICAL SOCIETY NEWS

H. Theodore Simon, M. D., Associate Editor.

The following Parish Medical Societies have elected officers for 1931:

Bienville Parish: President, Dr. R. C. Ferguson, Arcadia; Vice-President, Dr. J. M. Mosley, Arcadia; Secretary-Treasurer, Dr. J. N. Blume, Arcadia; Delegate, Dr. O. L. Kidd, Gibsland; Alternate, Dr. C. C. Allums, Ringgold.

East Baton Rouge Parish: President, Dr. Rufus Jackson, Baton Rouge; Vice-President, Dr. T. C. Foreman, Baton Rouge; Secretary-Treasurer, Dr. W. H. Pipes, Baton Rouge; Delegates, Dr. R. G. McMahon, Dr. H. W. A. Lee, and Dr. Rufus Jackson, all of Baton Rouge.

East and West Feliciana Bi-Parish: President, Dr. J. B. Sewell, St. Francisville; Vice-President, Dr. S. L. Shaw, Clinton; Secretary-Treasurer, Dr. E. M. Toler, Clinton; Delegate, Dr. Glen J. Smith, Jackson; Alternate, Dr. E. M. Robards, Jackson.

Jackson-Lincoln Bi-Parish: President, Dr. J. J. Bennett, Ruston; Vice-President from Lincoln Parish, Dr. D. S. Calhoun, Ruston; Vice-President from Jackson Parish, Dr. W. M. McBride, Hodge; Secretary-Treasurer, Dr. W. S. Rutledge, Ruston; Delegate, Dr. M. T. Green, Ruston; Alternate, Dr. W. M. McBride, Hodge.

Pointe-Coupee Parish: President, Dr. Jos. O. St. Dizier, Walls; Vice-President, Dr. J. F. Cazayoux, New Roads; Secretary-Treasurer, Dr. F. F. Rougon, New Roads; Delegate, Dr. R. McG. Carruth, New Roads; Alternate, Dr. J. O. St. Dizier, Walls.

Rapides Parish: President, Dr. G. C. Antony, Alexandria; First Vice-President, Dr. D. K. Texada, Alexandria; Second Vice-President, Dr. W. L. Murrell, Alexandria; Secretary-Treasurer, Dr. E. Weiner, Alexandria; Delegates, Dr. R. O. Simmons, Dr. J. H. Landrum, both of Alexandria, Dr. E. DeNux, Echo; Alternates, Dr. J. N. Thomas, Alexandria; Dr. J. W. Phillip, Boyce; Dr. J. A. Packer, Alexandia.

Washington Parish: President, Dr. D. A. Berwick, Bogalusa; Vice-President, Dr. H. V. Jones, Bogalusa; Secretary-Treasurer, Dr. Frank A. Williams, Franklinton; Delegate, Dr. J. W. Pafford, Bogalusa.

Webster Parish: President, Dr. C. T. Deloach, Sarpeta; Vice-President, Dr. S. M. Richardson, Minden; Secretary-Treasurer, Dr. W. McDade, Minden; Delegate, Dr. S. F. Martin, Minden; Alternate, Dr. John Pugh, Cotton Valley.

ASCENSION PARISH MEDICAL SOCIETY.

A meeting of the Ascension Parish Medical Society was held at the Elks' Home in Donaldsonville, La., at 11 A. M. Sunday, December 14, 1930, with the following members present: Drs. T. H. Hanson, D. T. Martin, E. S. Keyes, D. C. Brumfield, G. H. St. Amant, and H. A. Folse. After call to order, roll call, and reading of minutes, the application for membership of Dr. Myer Epstein of Gonzales, La., was voted upon favorably.

Dr. D. T. Martin, Parish Health Officer, reported that serums necessary for the treatment of the indigent sick were available through the Parish Board of Health. Dues of $7.00 for the State Society were paid by all members present. The Society collected no local dues for the year 1931.

Officers elected at this meeting were: Dr. H. A. Folse, President; Dr. C. D. Simmons, Vice-President, and Dr. D. C. Brumfield, Secretary-Treasurer; Dr. D. T. Martin, delegate to Louisiana State Medical Society meeting April, 1931; Dr. T. H. Hanson, Alternate.

EAST AND WEST FELICIANA BI-PARISH MEETING.

The Bi-Parish Medical Society met in the dining room of the Rist Hotel, December 3, 1930. At the close of the elegant banquet, Mrs. August Rist was presented to the assembled physicians by the Secretary, and words of felicitation were spoken to Mrs. Rist by Dr. Rufus Jackson on the feast she had prepared and served.

The scientific program consisted of a paper and clinical patient on Myositis Fibrosa by Dr. E. M. Toler of Clinton. A helpful and instructive discourse on Accessory Sinusitis, Nonpurulent, by Dr. Rufus Jackson of Baton Rouge. Last, but not least, came the enlightening address on Charity Hospital by the Superintendent, Dr. Arthur Vidrine of New Orleans.

Officers for 1931: Dr. J. B. Sewell, President; Dr. S. L. Shaw, Vice-President; Dr. E. M. Toler, Secretary-Treasurer; Delegate to the Louisiana State Medical Society Meeting, Dr. Glen J. Smith; Alternate, Dr. E. M. Robards.

Physicians present: Drs. Smith, Vidrine, Cook, Shaw, Jackson, Wilkinson, Levert, Lea, Daniel, Robards, Norris, Miller, Sewell, C. S. and E. M. Toler.

Glen J. Smith, President.
E. M. Toler, Secretary.

WOMAN'S AUXILIARY OF THE OUACHITA PARISH MEDICAL SOCIETY.

The physicians' wives of Ouachita Parish have established a Woman's Auxiliary, which will meet once a month at the Business and Professional Women's Club.

A course of study will feature the meetings in the future, interspersed with social affairs, the nature of which will be discussed and voted upon at the next meeting. The most important feature of yesterday's meeting was the election of officers as follows: Mrs. J. B. Vaughan, President; Mrs. Armand McHenry, First Vice-President; Mrs. J. Q. Graves, Second Vice-President; Mrs. C. P. Grey, Recording Secretary; Mrs. F. C. Bennett, Corresponding Secretary; Mrs. E. R. Yancey, Publicity Secretary; Mrs. R. W. O'Donnell, Treasurer; Mrs. J. E. Walsworth, Parliamentarian.

Present at the initial meeting were: Mrs. A. E. Fisher, Mrs. D. M. Moore, Mrs. John Williams, Mrs. John Snelling, Mrs. E. R. Yancey, Mrs. J. B. Vaughan, Mrs. C. P. Grey, Mrs. J. Q. Graves, Mrs. H. Guerriero, Mrs. J. Prather, Mrs. E. Powell, Mrs. R. M. McKoin, Mrs. Peters, Mrs. R. W. O'Donnell, Mrs. Armand McHenry, Mrs. J. W. Murphy, Mrs. C. H. Hill, Mrs. W. M. Johnson, Mrs. Clifford Johnson, Mrs. I. Wolff, Mrs. Perot, and Mrs. J. P. Brown.

HEALTH OF NEW ORLEANS.

The Bureau of Census of the Department of Commerce of the Division of Vital Statistics reported that during the week ending November 15 there occurred in the City of New Orleans 159 deaths, 93 of whom were white and 66 colored, giving a death rate of 18.1, contrasting with the corresponding week of a year ago when the death rate was 15.5. The next week ending November 22 there were 160 deaths in the city, the same proportion between the white and the colored, giving a death rate of 18.2, whereas in the corresponding week of 1929 the death rate was 16. The report was not received for the week ending November 29, but for the week ending December 6 it was found that the death rate had fallen slightly, there being 152 deaths, 88 among the white population and 64 in the colored, with a death rate of 17.3, whereas in the corresponding week of 1929 there was 179 deaths and the rate was 20.7.

CIVIL SERVICE EXAMINATIONS.

The United States Civil Service Commission announces the following named open competitive examinations: Medical Officer, Associate Medical Officer, Assistant Medical Officer (General Medicine and Surgery).

Applications for the above-named positions will be rated as received by the U. S. Civil Service Commission at Washington, D. C., until June 30, 1931. These examinations are to fill vacancies in the Department Service, Veterans' Bureau, Public Health Service, Indian Service, Coast and Geodetic Survey, and Panama Canal Service. Competitors will not be required to report for examination at any place, but will be rated on their education and training, and on their experience.

Full information may be obtained from the United States Civil Service Commission, Washington, D. C., or from the Secretary of the United States Civil Service Board of Examiners at the Post Office or Custom House in any city.

Surgeon O. E. Denny was directed by the Surgeon General to proceed from Carville, La., to Washington, D. C., and return, for conference at the Bureau.

A NEW LOUISIANA MEDICAL SCHOOL.

After going to press announcement has been made that there will be established a new medical department in the Louisiana State University. This news is of so much importance to the medical profession of Louisiana that we feel obliged to run it in the Journal despite the fact that it can only be put in as an addition to this column.

Governor Long in his announcement said that the first two years will be conducted at Baton Rouge; the last two years will be in New Orleans in the Charity Hospital. The general plan is that the new school will start to function next fall. There will be required of the students a year's service as an interne before receiving their degree. The Charity Hospital will have ample space not only for the new school but also for the Tulane Medical School. The Governor announces that it is his plan to give to the prospective medical students of Louisiana of Louisiana an education, which will be within their financial resources and which will increase the number of physicians practicing in the State, more particularly in the country districts. The new school will be a splendid addition to the medical activities of the State, and should be welcomed by everyone in the profession.

MISSISSIPPI STATE MEDICAL ASSOCIATION NEWS

L. S. Lippincott, Editor

H. L. Rush, Associate Editor *D. W. Jones, Associate Editor*

FROM OUR PRESIDENT
COUNTY SECRETARIES' REPORTS

If we did not get our dues paid in December, and, of course, some of us forgot about it, we had better get busy now and send them in, because the Council cannot defend any suits for malpractice that may be brought against us during the time, after January first, if the are unpaid. Of course, no one expects to be sued, but it is astonishing how many are and we, ourselves, may be the next ones.

Remember, too, that the County Secretaries' reports should be in the hands of the State Secretary "by February first;" and that unless they are, the delinquent society is automatically suspended until the report is sent. This kind of suspension is usually not particularly painful and doesn't leave any bad after-effects, but it isn't to the credit of anyone to be suspended for non-payment of dues, even though Dr. Dye, out of the kindness of his heart, doesn't make the bad news present. And it is easier to pay dues in January than in February, because, even though the Christmas bills may be staggering, taxes seem worse.

The County Secretary is the hardest worker in the Society and all he gets in the way of reward is the knowledge that he is doing a job for organized medicine, plus an occasional word of thanks and frequently some criticism. We should endeavor to make his work as light as possible and we can best do this by "coming across" promptly. Do it now!

Best wishes for a happy New Year!

E. F. Howard.

A VERY HAPPY AND PROSPEROUS NEW YEAR TO EVERYONE

is the sincere wish of your editors. Those whom you have chosen to represent you on the staff of "our Journal" are working conscientiously to make the Mississippi section worthy of the great profession they serve. They take this opportunity to gratefully acknowledge the fine spirit of co-operation and kind words of encouragement that have been so generously given. They realize fully that to carry on the work to your satisfaction and as you would desire, it is very necessary that every member of the Mississippi State Medical Association take an active and constructive interest in every month's issue of the Journal. They know that unless you let us know what you are doing, what your colleagues are doing, what your hospitals are doing, what your County Medical Societies are doing, many items of interest to the medical profession of the State will go unchronicled. It is the wish of your editors to make the Mississippi section a complete history of the profession from month to month, so that in the years to come, there may be a reliable record for our successors. Many times, had we such a record of the past, it would now be invaluable.

Will you not today and now make a great *New Year's resolution* to inform your editors at least once a month of the medical happenings of your community.

We are here to serve you. Will you help?

SOUTH MISSISSIPPI MEDICAL SOCIETY.

At the quarterly meeting of the South Mississippi Medical Society held at Laurel on December 12, Dr. W. S. Harper, Laurel, was elected President; Dr. W. N. Blount, Laurel, first Vice-President; Dr. Lowery, Hattiesburg, second Vice-President; Dr. J. H. Newcomb Richton, Secretary-Treasurer (re-elected). The new officers do not take office until June.

The program included addresses and papers by Drs. J. G. Gardner, Columbia; C. C. Hightower, Hattiesburg; D. W. Jones, Jackson; Joseph E. Green, Richton; C. C. Buchanan, Hattiesburg; T. E. Wilson, Jackson; J. P. Culpepper, Hattiesburg; and T. R. Beech, Ellisville.

The next meeting of the Society will be held in Hattiesburg in March.

NEW REGISTRATIONS.

At a meeting of the Mississippi State Board of Health on December 11, licenses to practice medicine in the State were granted through reciprocity to the following: Drs. Crit Harris, Louisville; Henry Grady Cook, Hattiesburg; Daniel Lester Hollis, Biloxi; Vinton Porter King, Hattiesburg; John K. Bullock, Jackson; Martin L. Smith, Hattiesburg; A. M. McCarthy, Electric Mills; Adolph Henriques, Pascagoula; and J. E. Hart (negro), Hub.

CENTRAL MEDICAL SOCIETY.

The Annual meeting of the Central Medical Society was held on December 16, in the Club Room of the Edwards Hotel, Jackson. After a banquet, the following scientific program was presented:

Granuloma Ingunale—Dr. John H. McLain, Jackson.

Foreign Bodies in the Lower Respiratory Tract and Esophagus with a General Review of the Subject of Broncho-Esophagoscopy—Dr. Richmond McKinney, Memphis.

The meeting closed with a business session and election of officers for 1931.

CRAWFORD CLINIC.

As an aid to the unemployed during the emergency period, a free medical and sugical clinic has been opened at the Crawford Clinic and South Mississippi Infirmary, to be operated in conjunction with all local welfare, civic, fraternal and church bodies.

The services of clinic physicians will be available to both white and colored patients. The entire lower floor of the clinic building has been made available for this work and a separate entrance has been provided. At present the clinic is in operation on Monday, Wednesday, and Friday afternoons from three to five o'clock. Should need develop for more frequent hours, changes will be made in the schedule.

No person will be admitted to the clinic for treatment who has not come through one of the established welfare agencies. Organizations sending patients will be required to purchase necessary medicines. Several drug stores have already agreed to fill prescriptions on a cost basis.

Mr. W. Hamilton Crawford, Superintendent, is in charge of the clinic.

DR. I. A. SHELBY.

Dr. J. Gould Gardner, Columbia, reports that following an illness of only a few days' duration, Dr. I. A. Shelby, Columbia, died December 10.

Dr. Shelby was 58 years of age and has practiced in Columbia for about 25 years. He is survived by his wife, two brothers, Mark Shelby of New Orleans, and Will Shelby of Port Gibson, and one sister, Mrs. Willis Maxwell, Port Gibson.

Mrs. L. L. Polk, Purvis, President of the Women's Auxiliary of the Mississippi State Medical Association, continues ill and is now at the South Mississippi Infirmary, Hattiesburg. Mrs. Polk has the best wishes of her many friends throughout the State for an early and complete recovery.

DE SOTO COUNTY MEDICAL SOCIETY.

Dr. A. J. Weissinger, reports as follows:

"I am sorry I have no medical news for you at this time, but will be glad to respond whenever anything of interest occurs in our society or otherwise. We are plodding along in the usual way up here in DeSoto."

NOTHEAST MISSISSIPPI THIRTEEN COUNTY MEDICAL SOCIETY.

A very pleasing courtesy was accorded to Dr. F. J. Underwood, Jackson, when the regular quarterly meeting of the Northeast Mississippi Thirteen County Medical Society, held at Aberdeen on December 16, was designated "Underwood Day."

The announcement of the meeting contains the following:

"This meeting is dedicated to Dr. F. J. Underwood, recently elected President of the Southern Medical Association, commemorating the honor that has justly fallen on a former Monroe County Doctor, a life long member of this Society, and the Executive head of the Mississippi State Board of Health."

The program included:

Invocation—Rev. R. E. McBlaine.

The X-Ray as an Aid in Differential Diagnosis—Dr. A. J. Stacy, Tupelo.

Discussion opened by Drs. J. R. Williams and R. M. Boyd.

President's Address—Dr. K. F. McRea, Corinth.

Hemophilia, with Case Report—Dr. W. M. Adams, Booneville.

Discussion opened by Drs. M. Q. Ewing and N. S. Dickson.

The Diagnosis and Treatment of Allergic Diseases—Dr. Ray M. Balyeat, Oklahoma City.

Discussion opened by Drs. W. H. Anderson and J. P. Henry.

The County or Community Hospital—Dr. R. B. Caldwell, Baldyn.

The Acute Ear—Dr. D. W. Hamrick, Corinth.

Discussion opened by Drs. G. G. Armstrong and D. E. Staton.

At the business session, officers were elected for the coming year. A banquet was served after the business session.

PIKE COUNTY MEDICAL SOCIETY.

Dr. I. E. Stennis, Secretary, Pike County Medical Society, reports as follows:

"The Pike County Medical Society met in the Palm Room, McColgan Hotel, McComb, December 4, at 7 P. M.

"There were only sixteen of the members present. No doubt the reduced attendance was due to the rainstorm that started in the afternoon and continued through the night.

"Roll-call and reading of the minutes of the last meeting. Unfinished business disposed of, under the head of which was the report of a committee appointed by the President at the November meeting to draft resolutions of respect for Dr. E. B. French, who died October 10.

"Other matters coming under the head of new business being disposed of, the Society went into the election of officers to serve during 1931.

"Dr. E. M. Givens, Summit, offered Dr. I. E. Stennis' name for nomination for President; nomination seconded by Dr. W. F. Cotton. Dr. Stennis was unanimously elected.

"Dr. B. J. Hewitt, McComb, nominated by Dr. E. M. Givens, for Vice-President; nomination seconded by Dr. W. O. Biggs, Osyka. Dr. Givens was unanimously elected.

"Dr. R. H. Brumfield, McComb, was nominated for Secretary by Dr. W. O. Biggs; nomination seconded by Dr. B. J. Hewitt. Dr. Brumfield was unanimously elected.

"Following the election of the new officers, President L. W. Brock made a very interesting talk to the members of the Society, appealing to the members to co-operate with the officers of the Society, and in that way to help keep the Pike County Medical Society on the 'Honor-Roll.' He said, in part, in order to do that, you must help the Secretary to keep his membership roll representing 100 per cent of practicing physicians of the County. You must also co-operate with him in arranging his programs for the monthly meetings. The more attractive his programs, the better attendance we will have at our monthly meetings. The larger our monthly attendance, the better the harmony and fine fellowship that will exist in our Society, which is one of the most, if not the most important feature of the Society.

"The scientific features of the program included the following: Paper by Dr. Thomas Purser, subject, Report of Case of Cancer of the Omentum. Discussion opened by Dr. L. W. Brock, followed by others. Discussion closed by Dr. Purser. Dr. T. E. Hewitt was on the program, but was unable to attend the meeting. Several clinical cases were reported by members of the Society, followed by general discussions."

PIKE COUNTY HEALTH DEPARTMENT.

Dr. Louis D. Dickerson, McComb, reports that Pike County has been assured a model health unit by the Board of Supervisors appropriating sufficient funds to match the State Board of Health and town appropriations. This brings to Pike County a model county health unit, the like of which are to be found only in Massachusetts and Tennessee. This unit has been brought about through the consideration of Dr. Underwood, who was influenced largely by a $2500.00 appropriation from Mr. Bradshaw, a former citizen of Summit. These funds make it possible for Pike County to participate in the benefits of the Commonwealth Fund of New York State.

The McComb City Hospital has added to its equipment a modern laboratory and solarium, with Miss Margaret Ann Supple of the University of Minnesota as technician and physiotherapist.

CLARKSDALE AND SIX COUNTIES MEDICAL SOCIETY.

Dr. D. V. Galloway, Clarksdale, Secretary of the Clarksdale and Six Counties Medical Society, reports that at the Fifty-seventh Semi-Annual Meeting of the Society, at Clarksdale, on November 5, it was voted to hold one session at each meeting instead of two, as heretofore, the meeting to begin at 6 P. M. with a business program, to be followed by a banquet and the scientific program.

The Society voted to hold a joint meeting with the Delta Medical Society, the date to be set at the March meeting.

The annual election of officers resulted as follows. President, Dr. W. S. Slaughter, Jonestown; Vice-President from Coahoma County, Dr. A. W. Rhyne, Coahoma; Vice-President from Bolivar County, Dr. H. L. Cockerham, Gunnison; Vice-President from Tallahatchie County, Dr. J. G. Backstrom, Tutwiler; Vice-President from Tunica County, Dr. H. L. Williams, Tunica; Vice-President from Quitman County, Dr. J. D. House, Sledge; Member of the Board of Censors, Dr. A. G. Everett (re-elected); Member of Committee on Medico-Legal Defense, Dr. J. W. Gray (re-elected); Secretary and Treasurer, Dr. D. V. Galloway, Clarksdale (re-elected).

LEAKE COUNTY MEDICAL SOCIETY.

Dr. W. S. Martin, President Leake County Medical Society, writes as follows:

"Leake County Medical Society met and reorganized on November 17, with eight members present. The members have agreed to meet bimonthly and to be punctual in attendance.

"On December 9 we will have a meeting and have invited a number of Jackson doctors to meet with us and after this meeting I will give you a report of same.

"About ten of 17 doctors in Leake County are members and the rest will not join anything."

December 3, 1930.

IN MEMORY OF
CHARLES THOMPSON CHAMBERLAIN, M. D.

On November 6, 1930, in the death of Dr. Charles T. Chamberlain the Homochitto Valley Medical Society lost one of its charter members.

He was a keen and untiring student of medicine, skillful in the practice of his profession and his untiring interest and broad charity knew no class or distinction. He is missed and mourned for by rich and poor, by white and colored, alike.

As a physician and as a soldier the memory of what he sacrificed for patients and for his country will be a lasting monument to him.

Be it resolved, That the Homochitto Valley Medical Society make known its sympathy to the bereaved family and that these resolutions be spread on the minutes of the Society and that they be published in the New Orleans Medical and Surgical Journal.

R. D. Sessions,
L. S. Gaudet,
J. S. Ullman, Chairman.

NORTHEAST MISSISSIPPI THIRTEEN COUNTIES MEDICAL SOCIETY.

REPORT OF COMMITTEE ON NECROLOGY.

We your Committee on Necrology, beg leave to report:

During 1930 death has claimed five from our ranks. Five physicians have gone to their reward and left to us fellow physicians the heritage of lives well spent in our calling. Their virtues attracted us; their fidelity inspired us, and their faithfulness to duty has challenged us to continue our best efforts to alleviate human suffering, cure and prevent disease and live a life worthy of emulation. "To live in the hearts of those we love is not to die." We rejoice that they have lived and labored amongst us and have upheld and represented the best in our profession.

As we enter the task of recording their names we tender on behalf of our society our sincere sympathy to all who are bereaved on account of their going.

DR. J. AUGUSTUS EVANS.

Dr. Evans was born June 13, 1861. He graduated from the Kentucky School of Medicine, Louisville, Ky., in 1885. He did post-graduate work at Tulane in 1897 and 1925. He was a member of the Houston Lodge, No. 67, F. & A. M., and of the Methodist Church.

He was beloved by every one in his territory. It could truly be said of him, he practiced medicine for the love of the profession, and the money consideration, if considered at all, was a second consideration. The poor never called him in vain. Verily it could be said of him, "The poor in his territory have medical attention."

As a civilian he stood for the highest and best in his community. He was always on the moral side of every question and championed every progressive movement in the community.

He died February 11, 1930, at Houston, Miss., where he spent his life.

DR. WILLIAM HOWARD MILLER.

He was born August 24, 1845, at Pontotoc, Mississippi. He attended the University of Louisiana and Bellevue Hospital College, 1870-73. He began practice in Okolona, Miss., in 1873.

Full of years and full of honors he has upheld the best traditions of our profession.

DR. J. S. VOYLES.

Born November 14, 1869, in Corinth, Mississippi, he lived there and practiced continuously until his death, May 20, last. He was graduated from Memphis Hospital Medical College in 1892. For many years he was Health Officer for the City of Corinth and local surgeon for both the Mobile & Ohio and Southern Railways. For a few months he located at Burnsville, but returned to his practice in Corinth.

As a man and physician, he enjoyed the confidence and esteem of the community in which he lived.

DR. CHARLES DANIEL SHIPP.

Charles Daniel Shipp, son of Mr. and Mrs. O. W. Shipp, deceased, was born near Big Creek, Miss., October 16, 1862. He received his literary training in the public schools of Calhoun County, and the University of Mississippi. He took his degree in medicine with the class of 1885 at the Memphis Hospital College of Medicine and began practicing in his home community and practiced continuously there till his death. He was married to Miss Nannie Forest of Kilmichael, Miss., on March 6, 1880. His wife preceded him to death, having died on May 9, 1923. His death occurred February 18, 1930, as a result of paralysis. He had five brothers, all of whom are dead except two, T. F. and S. T. Shipp, of Big Creek; he also had one sister, Mrs. G. W. Pullen, who is dead. Two children survive him, J. Neely Shipp, and Mrs. Elliott Norman, both of Big Creek. He was an efficient and successful physician, loved by his professional colleagues and patrons; kind and patient in the sick room and lenient in his fees. He was above the average in intellect and took considerable interest in political affairs as also in the civic activities in his community.

MISSISSIPPI STATE BOARD OF HEALTH.

The Mississippi State Board of Health conducted a Maternity Institute for public health nurses at Jackson on December 8 and 9. Classes were in charge of Miss Anita Jones, R. N., of the Maternity Center, New York City.

The Eighteenth Annual Conference of Health Officers and Sanitary Inspectors was held in the Convention Hall, Robert Lee Hotel, Jackson, December 11 and 12. The program was published last month.

NORTH MISSISSIPPI SIX COUNTY MEDICAL SOCIETY.

The program announced for the meeting of the North Mississippi Six County Medical Society for

November 19, at New Albany, was as follows:

Invocation—Rev. Dorsey.

The Future Role of the Family Physician in Organized Medicine.—Dr. W. H. Anderson, Booneville.

Amebic Dysentery—Dr. H. J. Gondolf, University.

Discussion opened by Drs. L. A. Barnett and S. E. Cooper.

Some Opthalmoscopic Problems of the General Practitioner—Dr. J. B. Stanford, Memphis.

Discussion opened by Drs. J. I. Mayfield and W. C. Lester.

Injection Treatment of Varicose Veins—Dr. G. A. Brown, Water Valley.

Discussion opened by Drs. C. M. Speck and G. H. Wood.

A Consideration of Deaths from Heart Disease—Dr. Whitman Rowland, Memphis.

Discussion opened by Drs. Ira B. Seale and W. W. Phillips.

BAPTIST HOSPITAL.

A regular meeting of the Staff of the Baptist Hospital, Jackson, was held on November 14, Dr. F. H. Hagaman, President, presiding.

Drs. R. C. O'Ferrall and W. L. Hughes were elected to the Staff and the name of Dr. L. B. Mosely was proposed for membership. Reports of interesting features of the meeting of the American College of Surgeons at Philadelphia were given by Drs. Wall, Armstrong, Dobson, and Hagaman.

A report of the meeting of the Inter-state Postgraduate Medical Association in Minneapolis was given by Drs. Neal and Hagaman.

JACKSON INFIRMARY.

November 12, 1930.

Meeting called to order by President at 7 P. M.

Minutes of previous meeting read and approved.

Two deaths during the previous month reported, one by Dr. Rehfeldt; a very unusual case, "Luetic Obstruction of the Pylorus and Other Complications."

Dr. Crisler spoke to the subject of the "Ideal Doctor." He stated that Robert E. Lee was his ideal of all men, mentioned the qualities of mind and heart necessary to be an ideal man.

Dr. Wilde reported an unusual case of "chronic blepharitis," parasitic in origin, and unyielding to the usual forms of treatment, finally cured by the use of xylol. He demonstrated the form of parasite responsible.

Dr. Hall reported a case under special case reports of "granuloma fungoides," stating that the disease occurs in three definite stages, not only very rare, but difficult to diagnose. Treatment is roentgenray.

Dr. Hughes reported "Impaction of Wisdom Teeth," simulating sinusitis in its symptomatology.

Those present were doctors Crisler, Hughes, Wilde, W. H. Watson of Pelahatchee, Hall, Rehfeldt, Walker, Moseley, Maxwell, Sims, Hand, Huggins, Howard, Adkins and Womack.

G. E. Adkins, Pres.
N. C. Womack, Sec'ty.

MISSISSIPPI'S FIRST STATE BOARD OF HEALTH.

The State Board of Health is making an effort to secure a photograph of each physician who was a member of the first State Board of Health, and will appreciate any information that will assist in securing these pictures.

The following was compiled from the minutes of the Board, dated April 7, 1877, and shows that the following were members of the first Board of Health:

President—Robert Kells, M. D., Jackson.
Secretary—Wirt Johnson, M. D., Jackson.

For the State-at-large (Appointed March 28, 1877)
W. M. Compton, M. D., Jackson, term six years.
D. L. Phares, M. D., Woodville, term four years.
F. W. Dancy, M. D., Holly Springs, term two years.

Appointed upon recommendation of the Mississippi State Medical Association on April 7, 1877:

Name	PostOffice	Term
First District—		
J. M. Taylor, M. D.	Corinth	Six years
A. G. Smythe, M. D.	Baldwyn	Two years
Second District—		
T. D. Isom, M. D.	Oxford	Four years
John Wright, M. D.	Sardis	Two years
Third District—		
E. W. Hughes, M. D.	Grenada	Four years
S. V. D. Hill, M. D.	Macon	Two years
Fourth District—		
A. H. Cage, M. D.	Canton	Two years
P. J. McCormick, M. D.	Yazoo City	Six years
Fifth District—		
Robert Kells, M. D.	Jackson	Six years
C. A. Rice, M. D.	Brandon	Six years
Sixth District—		
R. G. Wharton, M. D.	Port Gibson	Four years
P. F. Whitehead, M. D.	Vicksburg	Four years

During 1878 (the year after the appointments were made) the great yellow fever scourge visited the state and four of the above-named members of the State Board of Health lost their lives. They were: Drs. E. W. Hughes, P. F. Whitehead, A. H. Cage, and W. M. Compton.

REPORT TUBERCULOSIS.

Dr. H. C. Ricks, Director, Bureau of Communicable Diseases, Mississippi State Board of Health, calls the attention of all physicians to the law which requires that all cases of tuberculosis shall be reported by name to the Secretary of the State

Board of Health. Under the present scheme of obtaining reports, such reports should be made through the County Health Officer in the same way that reports of other communicable diseases for which clinical report cards are required are made.

PREVENTABLE DISEASES.

The report of the Bureau of Communicable Diseases of the Mississippi State Board of Health for the month of October, 1930, shows 128 cases of typhoid fever, 4 cases of small-pox, and 301 cases of diphtheria. All of these diseases were preventable!

ISSAQUENA-SHARKEY-WARREN COUNTIES MEDICAL SOCIETY.

The regular monthly and annual meeting of the Issaquent-Sharkey-Warren Counties Medical Society, in joint session with the Fifth District Medical Society of Louisiana, was held at Vicksburg on December 9 with 112 in attendance.

Guests of the Society included physicians from five states.

During the banquet the following distinguished guests were introduced and made brief talks: Dr. F. J. Underwood, Jackson, President, Southern Medical Association; Dr. William Gross Harrison, Birmingham, Vice-President, Southern Medical Association and President, Alabama State Medical Association; Dr. Hermann B. Gessner, New Orleans, President Louisiana State Medical Society; Dr. E. F. Howard, Vicksburg, President, Mississippi State Medical Association; Dr. John C. Culley, Oxford, President-elect, Mississippi State Medical Association; Dr. C. L. Mengis, Sterlington, La., President, Fifth District Medical Society; Dr. F. P. Rizzo, Monroe, Secretary, Fifth District Medical Society; Dr. C. L. Mull, Oxford, Dean, Medical School, University of Mississippi; Dr. Daniel J. Williams, Gulfport, President, Council, Mississippi State Medical Association; Dr. D. W. Jones, Jackson, Councilor of Fifth District, Secretary of Council, Mississippi State Medical Association.

The scientific program included addresses as follows:

The Occurrence of Luetic Nephritis in Infancy and Childhood.—Dr. W. W. Butterworth, New Orleans.

The Anatomy and Surgical Treatment of Undescended Testicle.—Dr. Virgil S. Counseller, Rochester, Minn.

Cardiac Pain; Characteristics and Significance. —Dr. James G. Carr, Chicago, Ill.

During the meeting, at the request of Dr. A. G. Payne, Greenville, President-elect, Tri-State Medical Association, it was announced that the next meeting of the Association would be held in Memphis about the middle of February. Letters and telegrams of regret for non-attendance were read from Drs. H. W. Kostmayer, New Orleans; M. Y. Dabney, Birmingham, Editor, Journal Southern Medical Association; J. H. Musser, New Orleans, Editor, New Orleans Medical and Surgical Journal; Dr. S. J. Couvillon, Moreauville, First Vice-President, Louisiana State Medical Society; Dr. C. A. Weiss, Baton Rouge, Louisiana, Second Vice-President, Louisiana State Medical Society; Dr. James S. McLester, Birmingham; Dr. E. E. Barlow, Dermott, Ark., President, Arkansas State Medical Association; Dr. C. P. Talbot, New Orleans, General Manager, New Orleans Medical and Surgical Journal; Dr. S. C. Barrow, President-elect, Louisiana State Medical Society, Shreveport.

The following officers for the year of 1931 were elected: President, Dr. J. B. Benton, Valley Park; Vice-President, Issaquena County, Dr. T. W. Huey, Grace; Vice-President, Sharkey County, Dr. H. S. Goodman, Cary; Vice-President, Warren County, Dr. F. M. Smith, Vicksburg; Secretary-Treasurer, Dr. L. S. Lippincott, Vicksburg; Member of Board of Censors for three years, Dr. L. J. Clark, Vicksburg; Member of Committee on Medical Defense, Dr. E. F. Howard, Vicksburg.

Dr. John Edwin Quidor, Vicksburg, was elected to membership in the Society.

A feature of the meeting was the presence of the President and Vice-President of the Southern Medical Association and three Presidents of State Associations.

VICKSBURG SANITARIUM.

The regular meeting of the Staff of the Vicksburg Sanitarium was held on December 11 with Dr. L. J. Clark, President, presiding.

After analysis of the work of the hospital for the month of November, special case reports were presented as follows:

Carcinoma of the Ovary.—Dr. G. M. Street.

Discussed by Drs. A. Street, L. S. Lippincott, S. W. Johnson, J. A. K. Birchett, Jr., and C. S. Clayton.

Intestinal Obstruction Complicated by Pregnancy.—Dr. J. A. K. Birchett, Jr.

Dr. A. Street presented a report of the recent meeting of the Southern Medical Association at Louisville.

Selected radiographic studies were shown and discussed as follows: Fracture of femur; fracture of tibia and fibula(2 cases); carcinoma of mediastinal glands, metastatic from carcinoma of breast; pulmonary tuberculosis, probably arrested; carcinoma of the rectum; inverted stomach.

Officers for the year of 1931 were elected as follows:

President, Dr. G. C. Jarratt; Vice-President, Dr. L. J. Clark; Secretary, Dr. L. S. Lippincott.

BOOK REVIEWS

Internal Medicine: By Sir Humphrey Rolleston, Bart., G. C. V. O., K. C. B., M. D., Hon. D. Sc. D. C. L., LL. D. New York, Paul B. Hober, Inc. 1930. Pp. 92.

Another one of the series of hand books of medical history edited by Krumbhaar, dealing in this instance with the story of internal medicine. Delightfully presented and written with decided charm, Rolleston's rather succinct and brief review of the history of internal medicine will well repay perusing by those interested in a narrative of the development of the science of medicine.

J. H. MUSSER, M. D.

Manual of Physiology for Students and Practitioners: By H. Willoughby Lyle, M. D., B. S. (Lond.), F. R. C. S. (Eng.) and David deSouza, M. D., D. Sc. (Lond.), F. R. C. P. (Lond.) 3rd ed. Oxford University Press. 1930. 3 pl., 138 fig. 803.

A third edition of a work, first published in 1911, and written from the point of view of giving the student of physiology in a book of convenient size the chief facts of physiology. This it does concisely and accurately. Such a book is not, however, a substitute for the text-books of physiology dealing as they do with the historical development of the facts of physiology as they result from observation, experiment and interpretation.

HENRY LAURENS, Ph. D.

Principles and Practice of Medicine: By Sir William Osler, M. D., F. R. S. & Thomas McCrae, M. D. 11th ed. New York. D. Appleton & Co. 1930.

Osler's and McCrae's "The Principles and Practice of Medicine" makes its appearance in the 11th Edition, and from the standpoint of its suitability as a text book for students, causes considerable discussion at this time. The present trend of using, as a text, one written by a multiplicity of experts in polyglot styles carries with it objections not occasioned by a treatise on the entire field of internal medicine which comes from the pen of one man. If we could conceive of an omniscient individual "who knew medicine" and who could present in one small volume what he thinks are the essentials, we would indeed have the ideal medical text book. In Osler's day when clinical medicine was unembellished and ungarnished by endless experimental aids and abettments and he, the Arch-Clinician was regarded as one whose word was law and whose opinion dare not be questioned, his book answered the need. However, in the past few years of devastating specialization and investigation with the attendant advances any single brain child is put to shame by the combined efforts of many.

Faithful to the senior author, the editor immortalizes his style so that it is difficult to tell which parts are written by whom and quotations from the literature of 1929 seem to flow from the pen of Sir William himself.

Considerable alterations or additions have been made to Undulant Fever, Diabetes Mellitus (including the use of Synthalin), Duodenitis, Cardiospasm (Phrenospasm), Hemorrhagic Nephritis, Nephrosis, Nephritis in general, Purpura (the various forms), Paroxysmal Cardiac Dyspnea, Infantile Splenic Anemia, Glosso-pharyngeal Neuralgia, and Compression Fractures of the Spine.

The following clinical gems have been added: Melioidosis, Uveoparatid Fever, *Bacillus faecalis alkaligenes* Infection, Exanthema Subitum, Poisoning from Radio-active Substances, Poisoning from Arseniuretted Hydrogen, Poisoning from Methyl Chloride, Hypoglycemia, Asbestosis, Pentosuria, Subacute Leukemia, Coronary Artery Aneurism, Macrogenitosomia Praecox, Myelophthisic Splenomegaly, Lipoid Histocytosis, Post-vaccinal Encephalitis, Encephalitis Periaxialis Diffusa, Centro-labor Cerebral Sclerosis, Osteitis Firbosa Cystica, Albers-Schonberg Disease, Spondylolisthesis, Defects in Membranous Bones with Exophthalmos and Diabetes Insipidus, Hypophyseal Cachexia, and Hemiballismus.

Despite the obvious objections to the book one can not deny that it is the best book of its type we know and it will probably enjoy a limited period of usefulness as a text. Whether it can be perpetuated ad infinitum, however, is very doubtful.

MAURICE SULLIVAN, M. D.

Diseases of the Skin: A Text-book for Practitioners and Students: By George Clinton Andrews, A. B., M. D. Philadelphia. W. B. Saunders Company, 1930. 998 illus. pp. 1091.

Dr. Andrews has divided his subject into thirty-six chapters and index.

The chapters on Roentgen-ray Therapy, Super-Soft X-Rays (w Rays, Grenz Rays), Radium Therapy, Ultra-Violet Light, and Surgical Diathermy are master pieces in themselves. The chapter on Syphilis is also well above par. The author has spared no expense or time in collecting illustrations, a very complete bibliography, and the last accepted opinion on Fungi, Tropical Diseases and Neoplasms. Dr. Andrews has learned from hard work and late study in American and European clinics what should be included in a text for Study, Reference and Practice. Here it is!

M. T. VAN STUDDIFORD, M. D.

Le Ditabète Sucré, Son Traitement: Par P. A. Carrié. Paris. G. Doin & Cie. 1930. 94 pp.

A concise, practical presentation of the problem of the treatment of the diabetic patient. It can be heartily recommended to the French reading members of the profession.

While the dominating influence of the French school (chiefly that of Marcel Labbé) is naturally apparent throughout, still generous recognition is given to the numerous valuable contributions, particularly by Americans and Germans. In this connection it may be urged that a proper bibliography or at least the addition of footnotes giving definite references would add greatly to the value of the work.

The author follows, in general, the views now generally accepted in America. He strives to render simple the instructions for determining the proper diet for any given patient. The reviewer is in hearty sympathy with this endeavor, and regrets that the author may have somewhat confused his readers by inserting alternate methods of calculation as suggested by various Americans. Insulin is apparently resorted to more reluctantly in France than in the United States. The author refers to and apparently employs methods of undernutrition, all of which have been given up in this country.

I. I. LEMANN, M. D.

The Pathology of Diabetes Mellitus: By Shields Warren, M. D. With a foreword by Elliott P. Joslin, M. D. Philadelphia. 1930. Lea & Febiger. 212 pp.

This study is based on the autopsies of 300 diabetics and 200 non-diabetics. The author begins by considering the historical development of the pancreatic theory of diabetes. He then takes up in turn the evidence, pro and con, of the involvement of the whole organ, the involvement of the islet tissue and the acinar tissue, and of the transition theory of the interrelation of islands and acini. He then points out, justly, that the cases of hyperinsulinism due to carcinoma of the islands of Langerhans reported in the last few years have served to clinch the truth of the insular hypothesis. His experience, however, has shown that "practically any lesion found in the pancreas of diabetic individuals either involving the island tissue, the acinar tissue or both can be duplicated in the pancreas of non-diabetics. However, the frequency of lesions of the islands is much less in non-diabetic individuals than in diabetics. Therefore, the occurrence of any of the generally accepted insular lesions may be considered as presumptive evidence of diabetes. However, without information of the previous clinical course or without studies of the glycogen distribution in other organs, the diagnosis of diabetes cannot be made. Interstitial pancreatitis occurs too frequently and too wide a range of disease processes to be considered a characteristic lesion of diabetes. Lipomatosis is probably more nearly related to the amount of stored fat in the body than it is to pathological changes in the pancreas." "In any case, the pathology of the pancreas in diabetes cannot be considered as static. As seen at any one time it represents a balance struck between the factors tending to produce degenerative changes of the islands and the regenerative powers of the pancreas." "The pancreas in diabetes is not simply the scarred field of an old battle ground, but is the actual field of conflict. It does not submit without a struggle to injury, but endeavors to regenerate."

Dr. Warren's work is not confined to the pathology of the pancreas. He has covered adequately the whole field of pathological changes occurring in the diabetic, and apparently related to the diabetic process. The chapter concerning the involvement of the spleen and the reticulo-endothelial system by the abnormal deposition of lipoid material therein is of particular interest. Diabetics have a disturbed fat metabolism and suffer more commonly from gall-stones than do non-diabetics. Naturally, the author has to discuss the old question of the relation of the gall-bladder disease and diabetes, and to consider whether diabetes or gall-bladder disease appeared first. He does not, however, come to any conclusion.

Of the greatest importance is the relationship of the disturbance of fat metabolism to the production of arteriosclerosis leading not only to gangrene of the extremities, but also to coronary occlusion and infarction of the heart.

Dr. Warren's book is a welcome and important addition to our knowledge of diabetes.

I. I. LEMANN, M. D.

The Mycoses of the Spleen: By Alexander George Gibson, M. D., F. R. C. P. New York, The Macmillan Co. 1930. Pp. 169.

This little volume of some 160 pages is a presentation of the investigations of Dr. Gibson in regard to the mycelial origin of certain splenomegalies. He believes that acholuric jaundice, splenic anemia and Banti's disease are infective in origin and that the infective agent is a streptothrix. This organism was obtained in most instances from the spleen. Gibson's work is corroborated mainly by the French school headed by Prof. Nanta. In this country and England spleen cultures failed to demonstrate the presence of mycotic organisms in the splenomegalies. While the book is well written and sounds quite convincing, it clearly does not settle the etiology of this group of diseases until much further confirmation is at hand. Whatever the ultimate result may be, it is safe to say that Dr. Gibson's book should stimulate further research in this interesting field.

RANDOLPH LYONS, M. D.

Warren's Handbook of Anatomy: By John Warren, M. D., and Robert M. Green, M. D. Cambridge, Harvard University Press. 1930. xiv+384 pp., 324 illus.

At the death of Doctor Warren, over two years ago, his projected topographical atlas of anatomy was left unfinished. Though the original plan of the work has been modified, the present volume embodies the extensive series of drawings which had been completed, accompanied by text written largely by Doctor Green. "This volume is intended to be an atlas, with a descriptive text designed to adapt it as a handbook for dissection and as a work of reference for the surgeon and practitioner rather than as a complete textbook of anatomy."

The arrangement of the illustrations and text is regional, with parts devoted successively to: head and neck; trunk; upper extremity; lower extremity; thoracis cavity; abdominal cavity; pelvic cavity; perineal region. The dissections are planned especially for the demonstration of important relationships, and on the whole the drawings illustrating them are of excellent workmanship.

HAROLD CUMMINS, Ph. D.

The Hair; Its Care, Diseases and Treatment: By W. J. O'Donovan, O. B. E., M. D. (Lond.), M. R. C. P. Philadelphia, P. Blakeston's Son & Co. Inc. 1930. Pp. 218.

In this book, facts are evaluated, ad seriatum, due prominence given to the most important: a compact presentation of the subject with practically no irrevalent matter.

The chapters pertaining to prophylaxis are excellent. T. A. MAXWELL, M. D.

Medical and Surgical Year Book: Physicians Hospital of Plattsburgh, Vol. 1, 1929. Plattsburgh, N. Y., Wm. H. Miner Foundation. 1930. pp. 322.

This year book deserves high commendation. Although its varied contents includes no articles of glaring originality there are several of unusual interest. Campbell reports a case of extensive coronary thrombosis, whose electrocardiograph tracing was normal and whose pulse until 36 hours before death was regular with good quality, and had a rate of 80 per minute. The symptom of cardiac pain was the important factor in making the diagnosis. The best and completest article is Parounagian's "Diagnosis of Congenital Syphilis and Its Role in the Etiology of Degenerative Disease." Dr. Walter Shiever makes a dedicatory address in appreciation of the memory of William Beaumont and traces the life history of that romantic figure, describing in fascinating fashion his ups and downs and the immortal Alexis St. Martin of gastric juice fame.

To the editor and not the authors, however, should go the most of the credit. The binding, arrangement, printing, photographic reproductions and charts are splendid.

MAURICE SULLIVAN, M. D.

PUBLICATIONS RECEIVED.

The Macmillan Company, New York: Treatment of Epilepsy, by Fritz B. Talbot, M. D. Protozoan Parasitism of the Alimentary Tract, Pathology, Diagnosis and Treatment, by Kenneth M. Lynch, M. D.

W. B. Saunders Company, Philadelphia, and London: Introduction to Medical Biometry and Statistics, by R. Pearl. A Primer for Diabetic Patients, by Russell M. Wilder, M. D.

C. V. Mosby Company, St. Louis: Operative Gynecology, by Harry Sturgeon Crossen, M. D., F. A. C. S., and Robert James Crossen, M. D. A Manual of Normal Physical Signs, by Wyndham B. Blanton, B. A., M. A., M. D., Second Edition. Medical Jurisprudence, by Elmer D. Brothers, B. S., LL.B., Third Edition.

Paul B. Hoeber, New York: Historic Artificial Limbs, by Vittorio Putti, M. D. Nosography: The Evolution of Clinical Medicine in Modern Times, by Knud Faber, M. D., LL.D., Second Edition.

Oxford University Press, New York: Textbook for Nurses, by E. W. Hey Groves, M. D., B. S., F. R. C. S.

The Rockefeller Foundation, New York: Methods and Problems of Medical Education (Eighteenth Series)

Lea & Febiger, Philadelphia: Progressive Medicine, a Quarterly Digest of Advances, Discoveries and Improvements in the Medical and Surgical Sciences, by Hobart Amory Hare, M. D., LL.D.

F. A. Davis Company, Philadelphia: Microbiology and Elementary Pathology, by Charles G. Sinclair, B. S., M. D. Intestinal Toxemia, by Anthony Bassler, M. D., F. A. C. P.

William Wood & Company, New York: Chronic Arthritis and Rheumatoid Affections, by Bernard Langdon Wyatt, M. D., F. A. C. P.

Williams & Wilkins Company, Baltimore: Observations on the Courses of Different Types of Bright's Disease and on the Resultant Changes in Renal Anatomy, by D. D. Van Slyke, Edgar Stillman, Eggert Moller, W. Ehrech, J. F. McIntosh, L. Leiter, E. M. Mackay, R. R. Hannon, N. S. Moore, Christopher Johnson.

Tennessee State Medical Association, Nashville, Tenn.: The Centennial History of the Tennessee State Medical Association, 1830-1930, by Philip M. Hamer.

Librairie Felix Alcan, Paris: Naissance, Vie Et Mort, Des Maladies Infectieuses, by Charles Nicolle.